



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

### Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

### About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>





**LANE**

**MEDICAL**

**LIBRARY**

**LEVI COOPER LANE FUND**











**THE**

**Science and Practice of Medicine.**

**VOLUME I.**





THE  
SCIENCE AND PRACTICE  
OF  
MEDICINE.

BY  
WILLIAM AITKEN, M.D., EDIN.,  
PROFESSOR OF PATHOLOGY IN THE ARMY MEDICAL SCHOOL.

SECOND AMERICAN, FROM THE FIFTH, ENLARGED AND CAREFULLY  
REVISED, LONDON EDITION,

ADOPTING THE NEW NOMENCLATURE OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

WITH LARGE ADDITIONS,

BY

MEREDITH CLYMER, M.D.,

EX-PROFESSOR OF THE INSTITUTES AND PRACTICE OF MEDICINE IN THE UNIVERSITY OF NEW YORK;  
FORMERLY PHYSICIAN TO THE PHILADELPHIA HOSPITAL; ETC. ETC.

IN TWO VOLUMES.

WITH A MAP, LITHOGRAPHIC PLATE, AND NUMEROUS ILLUSTRATIONS  
ON WOOD.

VOL. I.



PHILADELPHIA:  
LINDSAY & BLAKISTON.  
1868.

W. J. B. A.

Entered, according to Act of Congress, in the year 1868,

By LINDSAY & BLAKISTON,

In the Clerk's Office of the District Court of the United States for the Eastern District of Pennsylvania.

SHERMAN & CO., PRINTERS.

## P R E F A C E

### TO THE SECOND AMERICAN EDITION.

THE first American edition of this work was out of print in little more than twelve months after publication. So rapid a sale may be accepted as an evidence of its appreciation by the profession of this country, and as a recognition of its claim to being a fair exposition of the Medical Science and Art of the day.

In the present edition the Editor has carefully revised his contributions, and added much new material. His additions are equal to about three hundred pages of the London edition. They will be chiefly found under the heads of: *Lardaceous Degeneration, Vaccination, Measles, Erysipelas, Typhoid, Relapsing, Yellow, and Malarial Fevers, Dysentery, Malignant Cholera, Malignant Pustule, Syphilis, Pathology of the Dietic Diseases, Scurvy, Parasitic Diseases, Rheumatism, Gout, Chronic Bright's Disease, Cancer, Tuberculosis, Diseases of the Nervous System, Diseases of the Heart and Lungs, the Sphygmograph, Pyæmia, Diseases of the Digestive Organs, Diseases of the Kidneys, and Diseases of the Cutaneous System.*

They also include *twenty-two new articles* upon subjects not treated of, or only incidentally mentioned, by the Author, namely:

Camp Measles,  
Spinal Symptoms in Typhoid Fever,  
Typho-Malarial Fever,  
Chronic Malarial Toxæmia,  
Chronic Camp Dysentery,  
Cholera Mòrbus,  
Cholera Infantum,  
Hereditary Syphilis,  
Gonorrhœal Rheumatism,  
Corpulence,  
Physical Diagnosis of the Diseases  
of the Brain and Spinal Cord,

Delirium of Inanition,  
Chronic Alcoholism,  
Epileptiform Neuralgia,  
Auscultation in Health and in Disease,  
Capillary Bronchitis,  
Plastic Bronchitis,  
Dilatation of the Bronchia,  
Fibroid Degeneration of the Lung,  
The Inoculation of Tubercle,  
Chronic Pyæmia,  
Syphilitic Disease of the Liver.

The subjects of Locomotor Ataxy, Glosso-Pharyngeal Paralysis, Aphasia, Dilatation of the Bronchia, the Sphygmograph and its tracings in disease, were introduced into this text-book by the Editor in the first American Edition (1866). They were first treated of by the Author in the Fifth English Edition (1868), and his articles on these disorders are chiefly condensed from those of the Editor, with the exception of the one on Dilatation of the Bronchia, which Dr. Aitken has abridged from Dr. T. G. Stewart's excellent article in the *Edinburgh Medical and Surgical Journal*, December, 1867.

The Editor's additions are printed in smaller type, and are thus designated: [ ].

M. C.

135 LEXINGTON AVENUE,

NEW YORK, September, 1868.

# P R E F A C E

## T O T H E F I F T H L O N D O N E D I T I O N .

---

A FAMOUS novel-writer of the day has recorded that “a Preface is a pleasant thing to write, whatever it may be to read.” To indorse the record, and to write a Preface for the *fifth* edition of this Text-book, is very gratifying to the author.

On the 6th of December, 1866 (twelve months after the publication of the *fourth* edition), the publishers wrote to the author, saying, “The state of the stock now renders it necessary for us to request you will prepare a *fifth* edition.”

In accordance with this request, the author has been engaged, during the past fifteen months, in a careful revisal of this work, and the result is that the *fifth* edition has increased in bulk upwards of 100 pages.

This increase implies many more considerable changes (which it is hoped are improvements), as well as additions, than can be made obvious in a preface. They consist, however, mainly, in—

*First.* The adoption and incorporation in the text of the “New Nomenclature of the Royal College of Physicians of London.” The plan and basis of this nomenclature and classification of diseases is stated at p. 174, vol. i, and a tabular view of the English portion of the Nomenclature of Diseases is substituted, at p. 175, vol. i, for the Nosology of Dr. Farr, which had been used in the previous editions. The reasons for this change are to be found in the account of the “Present State and Aim of Nosology,” at p. 171, vol. i.

*Second.* The new *English Nomenclature* of the College has been adopted throughout the text. The *definitions* of diseases, and the *foreign equivalents* for their English names, have been incorporated where the several diseases are described.

The author is indebted to the kindness of Dr. Sibson for the privilege of an early copy of the new *Nomenclature* of the College, as it passed through the press, with a view to its being used in this edition.

*Third.* The subjects of *Malignant Cholera*, of *Paralysis*, of *Epidemic Cerebro-Spinal Meningitis*, and of *Intestinal Obstruction*, have been entirely re-written.

*Fourth.* The subjects of *Progressive Locomotor Ataxy*, *Progressive Muscular Atrophy*, *Glosso-laryngeal Paralysis*, *Aphasia*, and *Dilatation of the Bronchial Tubes*, the application of the *Sphygmograph*, and its tracings in diseases, where it has been of use, are subjects considered for the first time in this Text-book.\*

As with previous editions, so with this one, the author has aimed at giving as fully and faithfully as he could the ideas and the views of the more advanced and able writers of the time, ever desirous that his Text-book should be a "representative book" of the Medical Science and Practice of the day, as actually understood and followed by the best men of our profession.

ROYAL VICTORIA HOSPITAL,  
NETLEY, April, 1868.

---

\* [This statement refers to the previous London editions of the work. All of the above subjects were fully treated of in the First American Edition "for the first time in this Text-book" by the Editor.—See *Preface* of the American Editor.]

## PREFACE TO THE FIRST EDITION.

---

IN the compilation of this HANDBOOK I have attempted to give a condensed view of the SCIENCE AND PRACTICE OF MEDICINE. It has also been my object to incorporate and connect the more recently established facts which illustrate the *Nature of Diseases* and their *Treatment* with the time-honored doctrines on which the Science of Medicine has been based.

While the greater portion of the volume is necessarily devoted to a consideration of the *Nature* and *Treatment* of individual diseases, a more comprehensive range of topics has been embraced, under the title of the SCIENCE OF MEDICINE, than it has hitherto been usual to include in text-books.

The introductory sections indicate the more important element of *General Pathology*; and those principles are shortly stated on which the more modern systems of *Nosology* have been founded since the time of Cullen.

The remainder of the volume, arranged in three divisions, treats, in the FIRST PART, of *Systematic Medicine, Nosology, or the Classification of Diseases*, and suggests that the classification of the Registrar-General of England should be adopted. This statistical nosology, originally proposed by Dr. William Farr, has been carefully discussed and revised at the recent meeting of the Statistical Congress held at Vienna, and a *nomenclature* substantially the same is proposed for adoption in all the States of Europe. The fatal cases are to be *registered* on a uniform plan. A definite *classification*, however, is still undetermined; but I am kindly informed by Dr. Farr that a *classification* nearly the same as the English one has been adopted in Bavaria, and is quietly making its way among practical men in Germany. The Austrians, also, as represented by Dr. Hebra, approve of the separation of *Zymotic* diseases from the others.

IN PART SECOND, under the head of the *Nature of Diseases, Special*





highest rank in the practical literature of this country ;” and his views regarding their nature are here preserved, commencing from page 185 of this Text-book. It was originally intended by my publishers to reproduce that Article, but I deemed it necessary entirely to re-write and re-model the whole, retaining the statements of facts, and such illustrations as appeared to be of sufficient importance. While I have collected information from every other available source, the limits of this volume prevent me doing more than simply stating at the end of each paragraph the name of the Author from whose writings the statements have been compiled. If I have correctly interpreted and stated the doctrines taught by the veteran laborers and original investigators in the fields of medical experience and research, the names of those of whose writings I have freely and largely availed myself will furnish a sufficient guarantee that the matter I have attempted to communicate is at least orthodox. Much valuable material I have also to acknowledge from anonymous contributors to the pages of the *Medical Journals*. For access to books and libraries I beg especially to express my thanks to Dr. Sieveking, Mr. Martin, Sir James Clark, Dr. Steele, and to the Library Committee of the Royal College of Surgeons. Lastly, my best thanks are due to Dr. Steele, Superintendent of Guy’s Hospital. Notwithstanding the unceasing demands upon his time which the onerous duties of his office entail upon him, he has kindly revised the sheets as they passed through the press ;—for their numerous imperfections I alone must bear the responsibility.

LONDON, 12th October, 1857.



# CONTENTS.

## PART I.

### TOPICS RELATIVE TO PATHOLOGY.

#### CHAPTER I.

	PAGE
OF MEDICINE AS A SCIENCE AND AS AN ART : ITS OBJECTS AND ITS EXTENT,	83
The Science of Medicine embraces a Study of—(1.) Physiology ; (2.) Pathology ; (3.) Therapeutics ; (4.) Hygiene,	84
The "Institutions" or "Theory of Medicine,"	84
A Study of Pathology the most useful Guide to the Student of Medicine,	84

#### CHAPTER II.

HOW THE PROVINCE OF PATHOLOGY IS MAPPED OUT, . . . . .	84
1. The Accurate Observation and Correct Registration of the Facts in Pathology,	84
2. Descriptive Pathology, General and Special,	84
History of Cases of Disease from their Origin to their End,	85
Statistics of Disease,	85
3. Speculative Pathology,	85
4. Pathology dictates the Maxims of Rational Practice,	85
Plan and Scope of this Text-book,	86

#### CHAPTER III.

RELATIVE NATURE OF THE TERMS "LIFE," "HEALTH," AND "DISEASE,"	86
Definition or Meaning of the Term "Disease,"	86
Meaning of the Terms "Life," and "Health,"	86
What ought to be embraced by the Definition of any Disease in particular,	87

#### CHAPTER IV.

HOW THE NATURE AND CAUSES OF DISEASES MAY BE ELUCIDATED, .	87
Subjects of Clinical Instruction,	87
Topics of Special Pathology,	87
Science of General Pathology,	87
Subjects for Investigation by the Student,	88

#### CHAPTER V.

OF MORBID PHENOMENA, SYMPTOMS, AND SIGNS OF DISEASE, . . .	88
Meanings attached to the Terms " <i>Sign</i> ," " <i>Symptom</i> ," and " <i>Diagnosis</i> ,"	88
How a " <i>Diagnosis</i> " is made,	88
How a " <i>Prognosis</i> " is given,	89
Significance of " <i>Pathognomonic Signs</i> ,"	89
Methodical Examination of Patients necessary,	89
Works recommended for Study,	40

CHAPTER VI.

	PAGE
MORBID ANATOMY AND PATHOLOGICAL HISTOLOGY: THE SPECIAL MEANS AND INSTRUMENTS BY WHICH THE NATURE OF DISEASES MAY BE INVESTIGATED, . . . . .	40
Definition and Scope of Morbid Anatomy and Histology, . . . . .	40
Morbid Anatomy is a Record of Facts, . . . . .	41
Its Relation to Pathology, . . . . .	41
Physiology must be the Basis of Sound Pathology, . . . . .	42
How the Science of Pathology is being Advanced, . . . . .	43
Characteristics of Medical Research in the Present Day, . . . . .	47
Use of Physical Aids to Diagnosis, . . . . .	48
Province of Morbid Anatomy, . . . . .	50

CHAPTER VII.

THE ELEMENTARY CONSTITUENTS OF LESIONS AS SHOWN BY MORBID ANATOMY AND OTHER MEANS OF RESEARCH, . . . . .	50
Means and Instruments of Research, . . . . .	50
Methods of Determining the Nature of Diseases, . . . . .	50
Various Forms of the constituent Elements of Disease, . . . . .	52
A. Morbid Elementary Products, . . . . .	52
B. Complex Vital Processes whose Phenomena more or less combined constitute Disease, . . . . .	54

CHAPTER VIII.

COMPLEX MORBID STATES, . . . . .	54
----------------------------------	----

SECTION I.—FEVER—*Pyrexia*.

Definition of Fever, . . . . .	54
Pathology of Fever, and Phenomena which constitutes the Febrile State, . . . . .	54
Abnormal Generation of Heat in Fever, . . . . .	55
Two Practical Points to be determined in Fever, . . . . .	55
1. The Amount of the Preternatural Heat, by accurate Measurement, . . . . .	55
2. The Amount of the Tissue-Change, represented by Excreta in relation to Body-Weight, . . . . .	56
The Sequence of Phenomena in Fever, . . . . .	56
The Usefulness of the Thermometer at the Bedside in the Diagnosis of Pyrexia, . . . . .	57
I. <i>The Instruments, Methods, and Practical Rules for observing and Recording the Temperature of the Human Body in Diseases where Fever is present,</i> . . . . .	58
Description of the Clinical Thermometers for Physicians made by Mr. Casella, 28 Hatton Garden, London, . . . . .	59
Directions for Using them, . . . . .	59
II. <i>Fluctuations of Temperature within the Limits of Health, and Correlation of the Animal Heat with the Pulse and the Respiration,</i> . . . . .	61
Collateral Circumstances which influence Animal Heat in Daily Life, . . . . .	63
III. <i>Ranges of Temperature in Disease indicated by the Thermometer alone,</i> . . . . .	64
Diagnosis of Disease based on continuous Thermometric Observation, . . . . .	64
IV. <i>Ranges of Temperature in Diseases where Fever is present, as related to the Amount of the Excreta,</i> . . . . .	71
(a.) Increase of Temperature with increased Elimination, . . . . .	78
(b.) Increase of Temperature with Products of Metamorphosis apparently diminished, . . . . .	74
(c.) Increase of Temperature with increased Metamorphosis, but with lessened Elimination, . . . . .	74
Local Lesions coincident with Suppression and Retention of the Excreta, . . . . .	75
Destruction of Tissue in the Febrile System, . . . . .	77
Excessive Retention of Water in the Febrile System, . . . . .	77
Condition of the Urine and of the Blood in Fever, . . . . .	78
Condition of the Pulmonary Excretion and of the Nervous System in Fever, . . . . .	78
Conditions which combine to produce the Complex Phenomena of Fever, . . . . .	79
Correlation of Pulse, Respiration, and Temperature, . . . . .	81

## SECTION II.—INFLAMMATION.

	PAGE
Definition of Inflammation, . . . . .	81
Pathology of Inflammation, . . . . .	82
Phenomena and Theory of the Inflammatory Process, . . . . .	82
Experiments and Observations of Professor Joseph Lister, . . . . .	82
I. <i>Altered Supply of Blood to the Part in Inflammation</i> , . . . . .	88
The Phenomena of "Stasis," . . . . .	87
II. <i>The Constitution of the Blood is altered in Inflammation as regards its adaptability to Nourish the Part</i> , . . . . .	87
Objects of more Extended Inquiry regarding Inflammation, . . . . .	89
The Causes of "Stasis" and of "Exudation," . . . . .	91
The Essential Nature of Inflammation, . . . . .	98
Products, Effects, or Events of Inflammation, . . . . .	94
Productive Effects of Inflammation—Inflammatory Effusions, or Exudations, . . . . .	95
The Serum of Inflammation—Serous Effusion, . . . . .	95
The Blood Effusions, or Extravasations of Inflammation, . . . . .	97
The Inflammatory Lymph, or Fibrine of Inflammation, . . . . .	98
Primordial Cell-Forms in Lymph, . . . . .	100
The "Compound Granule-Cell," or "Exudation-Corpuscle," . . . . .	101
The Nature and Growth of Pus-Cells, . . . . .	108
Formation of Pus—Suppuration, . . . . .	104
Parenchymatous and Secretory Inflammation, . . . . .	107
Events which Accompany or Follow Inflammation, . . . . .	108
Softening or Diminished Cohesion of Tissue, . . . . .	108
Ulceration, its Nature and Mode of Operation, . . . . .	108
Granulation, its Nature and the Mode of Operation through which a Wound or Sore is Healed by it, . . . . .	109
Mortification, its Nature and Occurrence in the <i>Humid</i> and <i>Dry</i> Form, . . . . .	110
Local and General Symptoms of Inflammation, . . . . .	111
I. Inflammatory Fever, . . . . .	111
Circumstances which modify Inflammatory Fever, . . . . .	118
II. Typhoid Fever as a Symptom of Inflammation, . . . . .	114
III. Hectic Fever as a Symptom of Inflammation, . . . . .	115

## SECTION III.—DEGENERATION OF TISSUE.

Definition and Pathology of Degeneration, . . . . .	117
(a) Fatty Degeneration and Histolysis, . . . . .	118
(b.) Mineral Degeneration, Calcification, or Petrification, . . . . .	120
(c.) Pigment Degeneration—Pigmentation—Blood-Crystals, . . . . .	121
(d.) Amyloid, Lardaceous, or Albuminoid Degeneration, . . . . .	124
Lardaceous Degeneration has been long known and described under other names, . . . . .	124
Albuminoid Nature of the so-called Amyloid Degeneration, . . . . .	126
Characteristic Test of the Existence of Amyloid Degeneration, . . . . .	127
General Characters and Anatomical Description of the Tissues which have undergone Amyloid Degeneration, . . . . .	129
Appearance of Lardaceous Degeneration in Organs, . . . . .	130
Elements of Tissue in which Lardaceous Degeneration has been Demonstrated, . . . . .	131
Origin of Amyloid Degeneration, and the Diseased States with which it has been found Associated, . . . . .	132
Clinical History, . . . . .	133
[Dr. Dickinson's Investigations], . . . . .	132
Signs or Symptoms associated with this Degeneration discoverable during Life, . . . . .	134
Dr. Stewart's Investigations, . . . . .	134
Amyloid Degeneration common amongst Soldiers, . . . . .	135

## CHAPTER IX.

TYPES OF DISEASE AND THEIR TENDENCY TO CHANGE, . . . . .	135
Changes of Type occur in Epidemic Fevers, . . . . .	135
Medicine shown to be a Productive Art, . . . . .	138

	PAGE
Types of Diseases are Modified by Complication with other Diseases, . . . . .	18
Types of Disease vary from time to time, . . . . .	141
Types of Inflammatory Fever changed, . . . . .	141
Elements of Congenital Degeneracy, . . . . .	148
Effects of Chronic Alcoholism, . . . . .	144
Varying Types of Disease prevail in Succession, . . . . .	145
View of the Subject entertained by Sir Thomas Watson, . . . . .	147

## CHAPTER X.

MODES BY WHICH DISEASES TERMINATE FATALLY, . . . . .	148
Mode of Death by extreme Old Age, . . . . .	148
by <i>Faint</i> or <i>Syncope</i> , . . . . .	149
by <i>Anæmia</i> , . . . . .	149
by <i>Asthenia</i> , . . . . .	149
by <i>Starvation</i> , . . . . .	150
by <i>Suffocation</i> , . . . . .	150
by <i>Coma</i> , . . . . .	151
"To Obviate the Tendency to Death" is a Fundamental Doctrine of Practice, . . . . .	151

## CHAPTER XI.

PRINCIPLES WHICH DICTATE THE TREATMENT OF THE COMPLEX MORBID PROCESSES, . . . . .	151
I. <i>As regards Fevers or the Febrile State</i> , . . . . .	151
How Fevers naturally Terminate favorably, . . . . .	151
Principles which ought to Guide the Treatment of Fever, . . . . .	152
II. <i>As regards Inflammation</i> , . . . . .	158
Principles which ought to Guide the Treatment of Inflammation, . . . . .	154
Antiphlogistic Treatment, . . . . .	154
Antiphlogistic Regimen, . . . . .	154
Antiphlogistic Remedies, . . . . .	154
General Rules relative to Bloodletting, . . . . .	156
The Influence of Purgatives in the Treatment of Inflammation, . . . . .	160
The Influence of Mercury, . . . . .	160
The Influence of Antimony, . . . . .	161
The Influence of Alkalies, . . . . .	161

---

## PART II.

### METHODICAL NOSOLOGY—SYSTEMATIC MEDICINE, OR THE DISTINCTIONS AND DEFINITIONS, THE NOMENCLATURE AND CLASSIFICATION OF DISEASES.

## CHAPTER I.

THE AIM AND OBJECTS OF NOSOLOGY, . . . . .	162
I. THE DEFINITIONS of Diseases, . . . . .	162
Principles on which Diseases may be Defined, . . . . .	163
II. THE NOMENCLATURE of Diseases, . . . . .	164
How Diseases are to be Named, . . . . .	165
III. THE CLASSIFICATION of Diseases, . . . . .	166
Usefulness of Classification, . . . . .	166
Principles on which Diseases have been Classified, . . . . .	167
The Aim of "Natural" Systems of Classification, . . . . .	170
Present State and Aim of Nosology, . . . . .	171
Dr. Farr's System of Nomenclature and Classification, . . . . .	171



## CONTENTS.

xvii

	PAGE
New Nomenclature adopted by the Royal College of Physicians of London, . . . . .	172
Plan of the Nomenclature of the Royal College of Physicians of London, . . . . .	174
Basis of the Classification adopted by the College of Physicians of London, . . . . .	174

## CHAPTER II.

<b>TABULAR VIEW OF THE "NEW NOMENCLATURE" ADOPTED BY THE ROYAL</b>	
<b>COLLEGE OF PHYSICIANS OF LONDON,</b>	<b>175</b>
<b>General Diseases—Section A.,</b>	<b>175</b>
Section B.,	176
<b>Arrangement of Local Diseases,</b>	<b>178</b>
<b>Diseases of the Nervous System,</b>	<b>178</b>
<b>Diseases and Injuries of the Eye,</b>	<b>179</b>
Ear,	180
<b>Diseases of the Nose,</b>	<b>181</b>
Circulatory System,	181
Absorbent System,	182
Ductless Glands,	182
Respiratory System,	183
Digestive System,	184
Urinary System,	187
Generative System,	188
<b>Affections connected with Pregnancy,</b>	<b>190</b>
<b>Diseases of the Female Breasts,</b>	<b>191</b>
Male Mammilla,	192
Organs of Locomotion,	192
Cellular Tissue,	193
Cutaneous System,	193
<b>Poisons,</b>	<b>195</b>
<b>Injuries (Operations omitted),</b>	<b>196</b>
<b>Human Parasites,</b>	<b>199</b>
<b>Congenital Malformations of the Fœtus,</b>	<b>200</b>

## PART III.

## THE NATURE OF DISEASES—SPECIAL PATHOLOGY AND THERAPEUTICS.

**CLASS I.—GENERAL DISEASES, HITHERTO CALLED ZYMOTIC.**

## CHAPTER I.

<b>GENERAL REMARKS ON THE PATHOLOGY OF GENERAL DISEASES, OR SO-CALLED ZYMOTIC DISEASES,</b>	<b>208</b>
<b>The Physiological Modes in which Poisons act Illustrate by Analogy the Nature of Zymotic Diseases,</b>	<b>204</b>
<b>Specific Actions of Poisons generally,</b>	<b>205</b>
<b>Specific Action of the Poisons which produce Zymotic Diseases,</b>	<b>208</b>
<b>Periods of Latency of Morbid Poisons,</b>	<b>209</b>
<b>Varying Effects of Morbid Poisons, and Peculiarities in their Modes of Action,</b>	<b>211</b>
<b>Mode in which Morbid Poisons Operate,</b>	<b>211</b>
<b>Morbid Poisons affect the Blood,</b>	<b>218</b>
<b>Deaths from Zymotic Diseases in this Country,</b>	<b>214</b>
<b>Four Orders Comprehended under the Class of Zymotic Diseases,</b>	<b>214</b>

CHAPTER II.

	PAGE
<b>PATHOLOGY OF THE MIASMATIC ORDER OF ZYMOTIC DISEASES, . . . . .</b>	<b>215</b>
The Sources of Miasmatic Diseases, . . . . .	215
Paludal Malarious Poison, . . . . .	215
Animal Malaria Poisons or Effluvia, . . . . .	216
Specific Disease-Poisons, . . . . .	217
Conditions Favorable to the Spread of Disease, . . . . .	217

CHAPTER III.

<b>ON THE NATURE OF ENDEMIC, EPIDEMIC, AND PANDEMIC INFLUENCES, .</b>	<b>218</b>
<i>Endemic</i> Influences, . . . . .	218
Propagation of some Diseases by Continuous Succession, . . . . .	219
Question as to the Spontaneous Origin of Diseases, . . . . .	221
Nature of <i>Epidemic</i> Influences, . . . . .	222
<i>Pandemic</i> Oscillations of Disease, . . . . .	224
Propagation of Typhoid Fever and Cholera, . . . . .	225

CHAPTER IV.

<b>MANAGEMENT OF EPIDEMICS; AND ON PROCEEDINGS WHICH ARE ADVIS- ABLE TO BE TAKEN IN PLACES ATTACKED OR THREATENED BY EPI- DEMIC DISEASES, . . . . .</b>	<b>226</b>
Principles which Guide the Management of Epidemics, . . . . .	227
Detail of Proceedings, . . . . .	227
Processes of Disinfection, . . . . .	231

CHAPTER V.

<b>DETAILED DESCRIPTION OF THE MIASMATIC ORDER OF ZYMOTIC DISEASES,</b>	<b>232</b>
---	------------

SECTION I.—ERUPTIVE FEVERS—*Exanthemata*.

<b>SMALL-POX—<i>Variola</i>, . . . . .</b>	<b>232</b>
Definition of Small-pox, . . . . .	232
Pathology of Small-pox, . . . . .	233
The Formation and Nature of the Small-pox Pustule, . . . . .	234
Varieties and Symptoms of Small-pox, . . . . .	236
1. Of Natural Small-pox and its Several Varieties, . . . . .	236
Symptoms of Small-pox without Eruption, . . . . .	236
Symptoms of the Distinct Small-pox, . . . . .	237
Period of Latency of Small-pox, . . . . .	237
Range of Temperature in Natural Small-pox, . . . . .	238
Diagram showing the Typical Range of Temperature in a Case of Natural Small-pox, . . . . .	238
Fever of Suppuration in Small-pox, . . . . .	239
Symptoms and Phenomena of Confluent Small-pox, . . . . .	240
2. Of the Inoculated Small-pox, . . . . .	242
Complications of Small-pox, and Special Morbid Tendencies, . . . . .	243
Pyogenic Fever after Small-pox, . . . . .	244
Sequelæ of Small-pox, . . . . .	244
Pustules on the Mucous Membrane in Cases of Small-pox, . . . . .	245
3. Of Small-pox after Vaccination—Varioloid, or Modified Small-pox, . . . . .	246
Symptoms, Course, and Complications of Modified Small-pox, . . . . .	246
Typical Range of Temperature in a Case of Modified Small-pox, . . . . .	247
Exhaustion of Susceptibility to the future Action of the Poison after an Attack of Small-pox, . . . . .	247
Coexistence of Small-pox with other Morbid States, . . . . .	247
Cause of Small-pox, . . . . .	248
Generation and Propagation of Small-pox, . . . . .	249
Prognosis and Causes of Death in Small-pox, . . . . .	249
Unfavorable Signs during the Course of Small-pox, . . . . .	250
Diagnosis of Small-pox, . . . . .	250
Remarkable Epochs in the History and Treatment of Small-pox, . . . . .	251

	PAGE
1. Therapeutic, Curative, or Sanative Treatment of Small-pox, . . . . .	252
Dietetic and General Treatment, . . . . .	252
Means adopted to prevent Pitting, . . . . .	255
2. The Prophylactic, Sanitary, or Preventive Treatment of Small-pox, . . . . .	255
Inoculation or Artificial Variolation, . . . . .	256
<b>COW-POX—<i>Vaccinia</i>, . . . . .</b>	257
Definition, Pathology, and Symptoms of Cow-pox, . . . . .	257
Communication of Human Small-pox to Cows, . . . . .	260
Small-pox and Cow-pox Identical in Nature, . . . . .	261
Vesicular Eruption of Horses as Sources of Variola, . . . . .	268
Symptoms of Natural Cow-pox, . . . . .	264
Primary Vaccine Lymph, . . . . .	265
<b>VACCINATION, . . . . .</b>	266
Legislative Enactments Relative to Vaccination, . . . . .	267
Nature of the Protection conferred by Vaccination, and the Evidence of its Existence, . . . . .	268
How the Protective Influence of Vaccination has been Impaired, . . . . .	273
Re-vaccination advisable from time to time, . . . . .	274
Protection Impaired by Imperfect Vaccination, . . . . .	276
[Results of Spurious Vaccination in the United States and Confederate Armies], . . . . .	276
The Operation of Vaccination, . . . . .	277
Signs of Successful Vaccination, . . . . .	279
Signs of Successful Re-vaccination, . . . . .	280
Characters of the Cicatrix after Vaccination, . . . . .	280
Means of Estimating the Efficiency of Vaccination, . . . . .	281
Selection of Lymph for Vaccination, . . . . .	282
<b>CHICKEN-POX—<i>Varicella</i>, . . . . .</b>	283
Definition and Pathology of Chicken-pox, . . . . .	283
Symptoms and Diagnosis of Chicken-pox, . . . . .	284
Treatment of Chicken-pox, . . . . .	285
<b>MILIARY FEVER—<i>Miliaria</i>, . . . . .</b>	285
Definition of Miliary Fever, . . . . .	285
Pathology and Symptoms of Miliary Fever, . . . . .	286
Treatment of Miliary Fever, . . . . .	287
<b>MEASLES—<i>Morbilli</i>, . . . . .</b>	287
Definition of Measles, . . . . .	287
Pathology of Measles, . . . . .	287
Diagram representing the Typical Range of Temperature in Measles, . . . . .	287
Symptoms of Measles, . . . . .	291
Characteristics of the Mild Form of Measles, . . . . .	291
Characteristics and Diagnosis of the more Severe Forms of Measles, . . . . .	292
[Camp Measles], . . . . .	293
Prognosis in Cases of Measles, . . . . .	294
Sequelæ, Causes, and Propagation of Measles, . . . . .	295
The Treatment of Measles, . . . . .	296
Treatment of the Pulmonary Complications in Measles, . . . . .	297
<b>SCARLET FEVER—<i>Febris rubra</i>, . . . . .</b>	299
Definition of Scarlet Fever, . . . . .	299
Pathology and Symptoms of Scarlet Fever in its Various Forms, . . . . .	299
Uræmic Phenomena and Desquamation in Scarlet Fever, . . . . .	301
Diagram of the Typical Range of Temperature in a Case of Scarlet Fever, . . . . .	302
Dropsey after Scarlet Fever, . . . . .	304
Varieties of Scarlet Fever, . . . . .	306
1. Simple Scarlet Fever, . . . . .	307
2. Anginose Scarlet Fever, . . . . .	307
Dangers in Anginose Scarlet Fever, . . . . .	308
3. Malignant Scarlet Fever, . . . . .	309
4. Latent Scarlet Fever, . . . . .	310
Sequelæ of Scarlet Fever, . . . . .	310
Diagnosis of Scarlet Fever, . . . . .	312
Cause and Propagation of Scarlet Fever, . . . . .	312

	PAGE
Prognosis and Treatment of Scarlet Fever, . . . . .	813
Dietetic and Preventive Treatment of Scarlet Fever, . . . . .	817
<b>HYBRID OF MEASLES AND SCARLET FEVER—<i>Rubeola</i>, . . . . .</b>	<b>818</b>
Definition and Pathology of <i>Rubeola</i> , . . . . .	818
Symptoms of the Hybrid Disease, . . . . .	819
The Eruptions in the Hybrid Disease, . . . . .	820
Diagnosis of the Hybrid Disease, . . . . .	821
Table showing the most Prominent Distinguishing Characters of Scarlet Fever, <i>Rubeola</i> , and Measles, . . . . .	822
Prognosis and Treatment of the Hybrid Disease, . . . . .	823
<b>DENGUE—<i>Denguis</i>, . . . . .</b>	<b>823</b>
Definition of Dengue, . . . . .	823
Pathology of Dengue, . . . . .	824
Symptoms of Dengue, . . . . .	824
Treatment of Dengue, . . . . .	825
<b>ERYSIPELAS—<i>Erysipelas</i>, . . . . .</b>	<b>825</b>
Definition and Pathology of Erysipelas, . . . . .	825
Symptoms of Erysipelas, . . . . .	827
Diagnosis of Erysipelas, . . . . .	827
Diagram showing the Typical Ranges of Temperature in a Case of Ery- sipelas, . . . . .	828
Local Symptoms of Erysipelas, . . . . .	829
Cause and Propagation of the Disease, . . . . .	831
Period of Latency in Erysipelas, . . . . .	832
Prognosis of Erysipelas, . . . . .	832
Treatment of Erysipelas, . . . . .	833
<b>THE PLAGUE—<i>Pestilentia</i>, . . . . .</b>	<b>835</b>
Definition of the Plague, . . . . .	835
Pathology and Historical Notice of the Plague, . . . . .	835
Symptoms and Phenomena of the Plague, . . . . .	838
Progress of the Bubo in Plague, . . . . .	838
Diagnosis and Cause of Plague, . . . . .	839
Modes of Propagation of the Plague, . . . . .	840
Treatment of the Disease, . . . . .	841
Preventive Treatment of Plague, . . . . .	841
Quarantine Establishments, . . . . .	841

## SECTION II.—THE CONTINUED FEVERS.

History of the Specific Distinction between Typhus and Typhoid Fever, . . . . .	842
<b>TYPHOID FEVER—<i>Febris enterica</i>, . . . . .</b>	<b>847</b>
Definition of Typhoid Fever, . . . . .	847
Pathology and Symptoms of Typhoid Fever, . . . . .	847
[Spinal Symptoms in Typhoid Fever], . . . . .	850
The Eruption in Typhoid Fever, . . . . .	853
Typhoid Fever in Children, . . . . .	855
The Temperature during Typhoid Fever, . . . . .	855
Typical Ranges of Temperature in Cases of <i>Typhoid</i> and <i>Typhus</i> Fever, contrasted with each other throughout their course, . . . . .	857
Duration of Attack and the Mode of Recovery, . . . . .	859
Events which Influence the Course of the Fever, . . . . .	860
Condition of the Urine in Typhoid Fever, . . . . .	861
Morbid Anatomy of the Lesions in Typhoid Fever, with special reference to the Phenomena and Progress of the Disease, . . . . .	863
Enumeration of the Lesions in Typhoid Fever, . . . . .	864
Anatomical Forms of the Intestinal Glands ( <i>foot-note</i> ), . . . . .	864
Modes by which Elimination of the Gland Lesion occurs, . . . . .	865
Characters of the Typhoid Ulcers, . . . . .	867
Occurrence of Intestinal Hemorrhage during Typhoid Fever, . . . . .	868
Lesions in the Mesenteric Glands, Spleen, and Lungs during Typhoid Fever, . . . . .	869
Growth of Tubercle during Typhoid Fever, . . . . .	870
Nature of the so-called " <i>Typhoid Deposit</i> ," . . . . .	872

	PAGE
Circumstances under which Death may Occur in Cases of Typhoid Fever,	872
[Prognosis of Typhoid Fever], . . . . .	878
[Diagnosis of Typhoid Fever], . . . . .	874
Origin and Propagation of Typhoid Fever, . . . . .	875
Preventive Measures, or Measures for Checking the Spread of Typhoid Fever, . . . . .	879
Specific Character of Typhoid Fever, . . . . .	880
Final Practical Indications for the Prevention of Typhoid Fever, . . . . .	882
Treatment of Enteric or Typhoid Fever, . . . . .	888
Treatment of the Diarrhœa of Typhoid Fever, . . . . .	884
Treatment of the Intestinal Affection, . . . . .	887
Treatment of Hemorrhages in Typhoid Fever, . . . . .	888
The Diet during Typhoid Fever, . . . . .	889
[Treatment during Convalescence], . . . . .	891
<b>TYPHUS FEVER—<i>Febris typhus</i>,</b> . . . . .	392
Definition and Nomenclature of Typhus Fever, . . . . .	392
Historical Notice of Typhus Fever, . . . . .	392
Causes which have produced Disease in the British Army ( <i>foot-note</i> ), . . . . .	394
Phenomena and Symptoms of Typhus Fever, . . . . .	395
The Eruption of Typhus Fever, . . . . .	396
Range of Temperature in Typhus Fever, . . . . .	399
Modes in which Typhus Fever may Terminate, . . . . .	400
Condition of the Blood in Typhus Fever, . . . . .	402
<i>Secondary Lesions and Complications in Typhus Fever,</i> . . . . .	402
1. Convulsions and Cerebral Affections, . . . . .	402
2. Secondary Pulmonic Complications, . . . . .	404
3. Gangrene of the Pulmonary Tissue, . . . . .	405
4. Secondary Cardiac Lesion, . . . . .	405
Prognosis in Typhus Fever, . . . . .	406
1. Combination of Symptoms and Phenomena which are of extremely Unfavorable Import, . . . . .	406
2. Combination of Symptoms or Phenomena which may be regarded as of Favorable Import, . . . . .	407
3. Modes of Fatal Termination, . . . . .	407
Morbid Anatomy, . . . . .	407
Treatment of Typhus Fever, . . . . .	407
General Indications for Treatment, . . . . .	408
Special Indications for Treatment, . . . . .	409
Indications for the Use of Stimulants in Typhus Fever, . . . . .	412
Necessity of very careful Nursing in cases of Typhus Fever, . . . . .	414
Treatment of the Head Affections, . . . . .	414
Origin and Propagation of Typhus Fever, . . . . .	416
Question of the Development of Typhus Fever <i>de novo</i> , . . . . .	416
Incubative Period of Typhus Fever, . . . . .	421
<b>RELAPSING FEVER—<i>Febris recidiva</i>,</b> . . . . .	422
Definition of Relapsing Fever, . . . . .	422
Pathology of Relapsing Fever, . . . . .	422
Historical Notice of Relapsing Fever, . . . . .	428
[Relapsing Fever in the United States], . . . . .	424
The Primary Paroxysm of Relapsing Fever, . . . . .	425
Phenomena of the "Crisis" in Relapsing Fever, . . . . .	427
Phenomena of the Relapse, . . . . .	427
Tendency to the Occurrence of Sudden Death, . . . . .	428
Duration of the Fever, and Prolonged Duration of Convalescence, . . . . .	428
Anatomical Characters, . . . . .	429
Sequelæ of Relapsing Fever, . . . . .	429
Treatment of Relapsing Fever, . . . . .	480
<b>FEBRICULA—<i>Febricula</i>,</b> . . . . .	481
Definition of Febricula, . . . . .	481
Pathology of Febricula, . . . . .	481
Range of Temperature in Febricula, . . . . .	482
Range of Temperature in a Case of Protracted Febricula, . . . . .	488
Treatment of Febricula, . . . . .	488
<b>SIMPLE CONTINUED FEVER—<i>Febris continua simplex</i>,</b> . . . . .	488

	PAGE
ANOMALOUS FORMS OF CONTINUED FEVER, . . . . .	484
Special Investigation required for " <i>Doubtful</i> " Cases, . . . . .	484
Necessity for the Accurate Investigation of Fever Cases, . . . . .	485
[SPECIFIC] YELLOW FEVER— <i>Febris flava</i> , . . . . .	486
Definition of Specific Yellow Fever, . . . . .	486
Pathology and Symptoms of Specific Yellow Fever, . . . . .	486
Professor W. C. Maclean's View of its Pathology, . . . . .	487
Phenomena of True or Specific Yellow Fever, . . . . .	488
History of the " <i>Eclair</i> " Epidemic, . . . . .	489
Period of Latency in Specific Yellow Fever, . . . . .	440
History of its Importation into St. Nazaire, . . . . .	441
Propagation beyond its usual Geographical Limits, . . . . .	442
Propagation of Yellow Fever by Fomites, . . . . .	443
[Yellow Fever in the Southern United States in 1862, 1864, 1867], . . . . .	444
Symptoms of Specific Yellow Fever, . . . . .	446
Clinical Phenomena of Specific Yellow Fever, . . . . .	447
<i>Types, Groups, or Forms</i> of Specific Yellow Fever, . . . . .	447
Prognosis in Specific Yellow Fever, . . . . .	451
Treatment of Specific Yellow Fever, . . . . .	451
[Prevention of Yellow Fever], . . . . .	454
 SECTION III.—THE LITTORAL, MALARIAL, OR PALUDAL FEVERS.	
[Malarious Fevers in the United States and Confederate Armies], . . . . .	456
General Pathology of the Malarious Poison, . . . . .	457
Persistent Pernicious Effects of Malaria, . . . . .	459
[Chronic Malarial Toxæmia, . . . . .	461
Morbid Anatomy of Malarial Toxæmia, . . . . .	461
Morphological Changes of the Blood in Malarial Fever], . . . . .	462
Causes and Modes of Propagation of Malarious Fever, . . . . .	464
Nature of Malarious Soils, . . . . .	465
Predisposing Causes of Malarious Fever, . . . . .	467
 AGUE OR INTERMITTENT FEVER— <i>Febris intermittens</i> , . . . . .	468
Definition of Intermittent Fever, . . . . .	468
Symptoms of Intermittent Fever, . . . . .	469
Phenomena of a Paroxysm of Ague, . . . . .	470
[Varieties in Types of Intermittent Fever], . . . . .	470
Primary Types of Intermittent Fever, . . . . .	471
[Types most prevalent in the United States and Confederate Armies], . . . . .	472
Range of Temperature in <i>Quotidian</i> Ague, . . . . .	473
Range of Temperature in <i>Tertian</i> Ague, . . . . .	474
Range of Temperature in <i>Quartan</i> Ague, . . . . .	474
Condition of the Urine in Ague, . . . . .	474
Treatment of Ague, . . . . .	475
[Treatment of Congestive Fever, . . . . .	478
Treatment of Chronic Malarial Toxæmia], . . . . .	479
 REMITTENT FEVER— <i>Febris remittens</i> , . . . . .	480
Definition and Symptoms of Remittent Fever, . . . . .	480
[Remittent Fever in the United States, . . . . .	483
Diagnosis between Remittent and Typhoid Fever, . . . . .	485
Malignant Congestive or Pernicious Remittent Fever, . . . . .	485
Anatomical Character of Remittent Fever], . . . . .	485
Treatment of Remittent Fever, . . . . .	486
Professor W. C. Maclean's Treatment of Remittent Fever, . . . . .	489
 MALARIOUS YELLOW FEVER— <i>Febris icterodes remittens</i> , . . . . .	491
Definition and Pathology of Malarious Yellow Fever, . . . . .	491
Conditions Favorable to the Development of Malarious Yellow Fever, . . . . .	493
Nature of the Soil in Malarious Districts, . . . . .	494
 [TYPHO-MALARIAL FEVER— <i>Chickahominy Fever, American Camp Fever</i> , . . . . .	495
Definition of Typho-Malarial Fever, . . . . .	495
History of Typho-Malarial Fever, . . . . .	495

	PAGE
Symptoms of Typho-Malarial Fever, . . . . .	495
Anatomical Characters of Typho-Malarial Fever, . . . . .	495
Treatment of Typho-Malarial Fever], . . . . .	496
SECTION IV.—MUCOUS FEVERS.	
<b>INFLUENZA</b> — <i>Catarrhus epidemicus</i> , . . . . .	496
Definition and Historical Notice of Influenza, . . . . .	496
Pathology of Influenza, . . . . .	497
Symptoms, Course, and Complications of Influenza, . . . . .	498
Causes and Modes of Propagation of Influenza, . . . . .	500
Prognosis of Influenza, . . . . .	501
Treatment of Influenza, . . . . .	501
<b>WHOOPIING-COUGH</b> — <i>Pertussis</i> , . . . . .	503
Definition, Pathology, and Morbid Anatomy of Whooping-cough, . . . . .	503
Symptoms of Whooping-cough, . . . . .	504
Complications of Whooping-cough, . . . . .	507
Diagnosis of Whooping-cough, . . . . .	508
Causes and Modes of Propagation of Whooping-cough, . . . . .	508
Period of Latency of Whooping-cough, . . . . .	509
Prognosis of Whooping-cough, . . . . .	509
Treatment of Whooping-cough, . . . . .	509
Dietetic and General Treatment of Whooping-cough, . . . . .	513
<b>MUMPS</b> — <i>Parotides</i> , . . . . .	513
Definition and Pathology of Mumps, . . . . .	513
Symptoms of Mumps, . . . . .	513
Treatment of Mumps, . . . . .	514
<b>DIPHTHERIA</b> — <i>Diphtheria</i> , . . . . .	514
Definition and Historical Notice of Diphtheria, . . . . .	514
Pathology and Morbid Anatomy of Diphtheria, . . . . .	515
Condition of the Urine in Diphtheria, . . . . .	516
Phenomena and Symptoms of Diphtheria, . . . . .	519
Varieties or Forms of Diphtheria, . . . . .	519
Prognosis and Sequelæ of Diphtheria, . . . . .	521
Paralysis after Diphtheria, . . . . .	521
Propagation of Diphtheria, . . . . .	522
Treatment of Diphtheria, . . . . .	523
<b>CROUP</b> — <i>Angina trachealis</i> , . . . . .	525
Definition, Pathology, and History of Croup, . . . . .	525
Phenomena of Croup, . . . . .	526
Symptoms and Course of Croup, . . . . .	529
Diagnosis and Modes of Propagation of Croup, . . . . .	530
Prognosis and Treatment of Croup, . . . . .	531
Question of Tracheotomy in Croup, . . . . .	533
[Statistics of Tracheotomy in Croup], . . . . .	534
<b>DYSENTERY</b> — <i>Dysenteria</i> , . . . . .	534
Definition of Dysentery, . . . . .	534
Historical Notice of Dysentery—Its Pathology and Morbid Anatomy, . . . . .	535
Prevalence and Mortality of Dysentery in Various Countries, . . . . .	536
Average Dates of Sickness and Mortality from Dysentery and Diarrhœa among European Troops in India, . . . . .	536
Various Forms of Dysentery, . . . . .	537
Acute Cases of Dysentery, . . . . .	537
Chronic Cases of Dysentery, . . . . .	538
Complex Cases of Dysentery, . . . . .	538
Anatomy of the Morbid Tissues in Acute Dysentery, . . . . .	538
Various Opinions as to the Nature of the Anatomical Lesions in Dysentery, . . . . .	539
Question relative to the Solitary Gland Lesion, . . . . .	540
Morbid Anatomy of Dysentery, . . . . .	541
Lesions in the Scorbutic Forms of Dysentery, . . . . .	543
Dr. Baly's Description of Dysentery, . . . . .	545





	PAGE
Evidences of Reaction, . . . . .	618
Chemical Changes undergone by the Body in the Progress of Cholera, .	619
[The Temperature in Malignant Cholera], . . . . .	621
Predisposing Causes of Malignant Cholera, . . . . .	626
Prognosis of Malignant Cholera, . . . . .	627
Treatment of Malignant Cholera, . . . . .	628
Prevention of Cholera, . . . . .	636
<b>[CHOLERA MORBUS—<i>Sporadic Cholera, Cholera Biliosa,</i></b> . . . . .	638
Definition of Cholera Morbus, . . . . .	638
History of Cholera Morbus, . . . . .	639
Nature and Pathogeny of Cholera Morbus, . . . . .	639
Symptoms of Cholera Morbus, . . . . .	639
Diagnosis and Treatment of Cholera Morbus, . . . . .	640
<b>CHOLERA INFANTUM—<i>Summer Complaint, Infantile Cholera,</i></b> . . . . .	641
Definition of Cholera Infantum, . . . . .	641
History of Cholera Infantum, . . . . .	641
Symptoms of Cholera Infantum, . . . . .	641
Causes and Nature of Cholera Infantum, . . . . .	642
Treatment of Cholera Infantum], . . . . .	644

## CHAPTER VI.

<b>PATHOLOGY OF THE ENTHETIC ORDER OF THE GENERAL OR (SO-CALLED)</b>	
<b>ZYMOTIC DISEASES,</b> . . . . .	646
General Disease arising from "Poisoned Wounds," . . . . .	646
Specific Effects of Implanted Morbid Poisons, . . . . .	646
Effects upon the Blood of the Poison of the Cobra di Capella, . . . . .	647
Changes which Implanted Morbid Poisons undergo, . . . . .	649
Varieties of Diseases produced by "Poisoned Wounds," . . . . .	650

## CHAPTER VII.

<b>DETAILED DESCRIPTION OF THE ENTHETIC ORDER OF ZYMOTIC DISEASES,</b>	651
<b>HYDROPHOBIA—<i>Rabies,</i></b> . . . . .	651
Definition of Hydrophobia, . . . . .	651
Pathology and Symptoms of Hydrophobia, . . . . .	651
Increase of the Poison in the System, . . . . .	653
Post-mortem Appearances in Hydrophobia, . . . . .	654
Symptoms of Hydrophobia, . . . . .	655
Remote Cause of Hydrophobia, . . . . .	657
Phenomena of Hydrophobia in the Dog, . . . . .	657
Communication of Hydrophobia, . . . . .	658
Diagnosis of Hydrophobia, . . . . .	659
Prognosis and Treatment of Hydrophobia, . . . . .	659
Preventive Treatment after Hydrophobic Inoculation, . . . . .	661
<b>GLANDERS—<i>Equinia,</i></b> . . . . .	661
Definition of Glanders, . . . . .	661
Pathology of Glanders, and Farcy in Man, . . . . .	662
Symptoms of Glanders, . . . . .	664
Cause of Glanders, . . . . .	665
Inoculation of Glanders, . . . . .	666
Period of Latency of Glanders, . . . . .	667
Diagnosis and Treatment of Glanders, . . . . .	667
Preventive Treatment of Glanders, . . . . .	668
<b>MALIGNANT PUSTULE (VESICLE?)—<i>Pustula pestifera,</i></b> . . . . .	668
Definition of Malignant Pustule, . . . . .	668
Pathology and Historical Notice of Malignant Pustule, . . . . .	668
Propagation of the Disease, . . . . .	669
Phenomena and Symptoms of the Malignant Vesicle, . . . . .	670
[Malignant (Edema of the Eyelids), . . . . .	671
[Anatomical Characters], . . . . .	672
Treatment of Malignant Pustule, . . . . .	672

	PAGE
SYPHILIS— <i>Syphilis</i> , . . . . .	673
Definition of Syphilis, . . . . .	673
Pathology and Morbid Anatomy of Syphilis, . . . . .	673
Prevalence of Syphilis in the Army, . . . . .	675
1. Nature of the Syphilitic Poison, . . . . .	677
Classification and Definition of the Forms of Syphilis, . . . . .	678
A. Primary Syphilis, . . . . .	678
B. Secondary Syphilis, . . . . .	678
C. Hereditary Syphilis, . . . . .	678
Nomenclature of Syphilis, . . . . .	678
Directions for Recording Syphilitic Cases, by Mr. Longmore ( <i>foot-note</i> ), . . . . .	678
2. Characters of Venereal Sores, . . . . .	679
Historical Identification of Venereal Sores, . . . . .	680
I. The Period and Doctrine of Hunter—the <i>Hunterian Chancre</i> , . . . . .	680
II. The Period and Doctrine of Ricord, . . . . .	681
III. The Present Period in the History of Syphilis, . . . . .	682
[Doctrines of the Unity and Plurality of the Syphilitic Virus ( <i>foot-note</i> )], . . . . .	682
Incubation of Syphilis, . . . . .	683
Contamination of the System, . . . . .	684
[Shakspeare's Description of the Effects of Constitutional Syphilis], . . . . .	684
Periods of Appearance of the Phenomena of Contamination, . . . . .	685
Cutaneous Affections, . . . . .	685
Affections of the Fauces, . . . . .	685
Second Attacks of Syphilis, . . . . .	685
Nature of the "Infecting Sore," . . . . .	686
Characters of " <i>Non-infecting</i> " Sores, . . . . .	687
Characters of <i>Herpes preputialis</i> ( <i>foot-note</i> ), . . . . .	688
[True Chancre], . . . . .	689
[Site of True Chancre], . . . . .	690
3. Vehicles or Media by which the Specific "Infecting" Virus may be Inoculated, . . . . .	691
4. Secondary Lesions and Morbid Anatomy of Syphilis, . . . . .	693
The <i>Gummata</i> of Syphilitic Lesions, . . . . .	694
Development and Course of the Syphilitic Node, . . . . .	696
Syphilitic Lesions in the Bones, . . . . .	696
Syphilitic Lesions in the Skin, . . . . .	697
Syphilitic Affections of the Nails, . . . . .	698
Syphilitic Lesions of the Heart, . . . . .	698
Syphilitic Lesions of the Brain, . . . . .	698
[Syphilitic Disorders of the Nervous System], . . . . .	699
Syphilitic Lesions of the Spinal Cord, . . . . .	702
Syphilitic Lesions of the Lungs, . . . . .	702
[Syphilitic Lesions of the Œsophagus, Stomach, Intestines, Rectum, and Diaphragm], . . . . .	703
Syphilitic Lesions of the Liver, . . . . .	703
[Syphilitic Lesions of the Spleen and Pancreas], . . . . .	703
[Syphilitic Lesions of the Kidneys], . . . . .	703
Syphilitic Lesions of the Testicles, . . . . .	704
[Syphilitic Lesions of the Muscles], . . . . .	704
Syphilitic Lesions of the Tongue, . . . . .	704
Hints for the Investigation and Description of Syphilitic Ulcers, . . . . .	705
Nature and History of the Doctrine of Syphilization, . . . . .	706
Process or Operation of Syphilization, . . . . .	707
Treatment of Syphilis, . . . . .	709
Mercury in the Cure of Syphilis, . . . . .	710
Present Position of Opinion regarding Mercury in Syphilis, . . . . .	710
[Propriety of giving Mercury in Syphilis], . . . . .	713
Iodide of Potassium in Syphilis, . . . . .	715
Treatment of the Suppurative Lesions in Syphilis, . . . . .	715
Mode of Administering Mercury in Syphilis, . . . . .	716
Vapor or Hot Air Bath in Syphilis, . . . . .	717
Preventive Treatment in Syphilis, . . . . .	718
[HEREDITARY SYPHILIS, . . . . .	718
Constitutional Syphilis transmissible in all its stages to children, . . . . .	719
Syphilis may affect the Fœtus at an early period, . . . . .	720

	PAGE
Coryza the Earliest and most Striking Symptom of Hereditary Syphilis, .	720
Appearance of the Teeth in Hereditary Syphilis, . . . . .	721
Affections of the Eye in Hereditary Syphilis, . . . . .	722
Phenomenal Table of the Stages and Symptoms of Inherited Syphilis], .	728

## CHAPTER VIII.

ON THE NATURE OF THE ACUTE SPECIFIC OR GENERAL DISEASES, . . . . .	724
Specific Characters, or "Specificity," of these Diseases, . . . . .	725
Origin of Acute Specific Diseases, . . . . .	726
Note by Mr. Simon, . . . . .	726
Defined Periods of Incubation, . . . . .	727
Defined Periods of Development, . . . . .	728
Defined Periods of Defervescence and Convalescence, . . . . .	728
An " <i>Active principle</i> " exists in some Specific Diseases, . . . . .	728
Modes of Propagation of Acute Specific Diseases, . . . . .	729

## CHAPTER IX.

PATHOLOGY OF THE DIETIC ORDER OF ZYMOTIC DISEASES, . . . . .	780
Effects of Food on the Animal Economy, . . . . .	781
[On the Food of Man in Relation to Useful Work], . . . . .	782
Certain Conditions necessary in the Adjustment of Dietaries, . . . . .	788
Nutritive Value of Foods in 100 Parts (PARKES), . . . . .	784
Dietaries and their Nutritive Values (LETHEBY), . . . . .	784
Nutritive Value of Foods (LETHEBY), . . . . .	785
Nutritive Value of Salted Meats, Tea, and Coffee, . . . . .	786
Effects of Overfeeding, . . . . .	787
[Corpulence, . . . . .	787
Symptoms of Corpulence, . . . . .	788
Pathogeny and Causes of Corpulence, . . . . .	788
Prognosis and Treatment of Corpulence, . . . . .	789
Mr. Banting's Management of Corpulence], . . . . .	740
Effects of Deficient Food, . . . . .	741
Death from Starvation, . . . . .	742
The "Truck System," . . . . .	744

## CHAPTER X.

DETAILED DESCRIPTION OF THE DIETIC ORDER OF ZYMOTIC DISEASES, . . . . .	745
SCURVY— <i>Scorbutus</i> , . . . . .	745
Definition of Scurvy, . . . . .	745
[Effects of Insufficient Food upon the Andersonville Prisoners], . . . . .	745
Pathology and Historical Notice of Scurvy, . . . . .	746
Morbid Anatomy and Pathogeny of Scurvy, . . . . .	747
[Scurvy in the United States and Confederate Armies], . . . . .	747
Drs. Christison's and Garrod's Theories relative to Scurvy, . . . . .	750
Symptoms of Scurvy, . . . . .	751
Diagnosis and Prognosis of Scurvy, . . . . .	755
Causes and Conditions under which Scorbutus is Developed, . . . . .	755
Treatment of Scurvy, . . . . .	759
Prevalence of Scurvy among Seamen in the Merchant Service ( <i>foot-note</i> ), . . . . .	760
Usefulness of Lemon-Juice in Scurvy, . . . . .	761
[Antiscorbutics], . . . . .	762
PURPURA— <i>Purpura</i> , . . . . .	764
Definition of Purpura, . . . . .	764
Pathology of Purpura, . . . . .	764
Symptoms of Purpura, . . . . .	765
Causes and Diagnosis of Purpura, . . . . .	766
Treatment of Purpura, . . . . .	767

	PAGE
ERGOTISM— <i>Morbus cerealis</i> , . . . . .	768
Definition of Ergotism, . . . . .	768
Historical Notice and Pathology of Ergotism, . . . . .	768
Phenomena of Ergotism, . . . . .	768
Symptoms of Ergotism, . . . . .	769
Treatment of Ergotism, . . . . .	770
 DELIRIUM TREMENS— <i>Delirium alcoholicum</i> , . . . . .	770
Definition of Delirium Tremens, . . . . .	770
Pathology and Historical Notice of Delirium Tremens, . . . . .	771
Effects of Alcohol as a Poison, . . . . .	772
Presence of Alcohol in the Blood and Fluids, . . . . .	772
Effects of Alcohol on the System, . . . . .	778
Symptoms and Course of Delirium Tremens, . . . . .	776
Diagnosis of Delirium Tremens, . . . . .	778
Prognosis of Delirium Tremens, . . . . .	778
Treatment of Delirium Tremens, . . . . .	779
 LEAD PALSY— <i>Paralysis ex plumbo</i> , . . . . .	781
Definition of Lead Palsy, . . . . .	781
Pathology of Lead Palsy, . . . . .	781
Diffusion of Lead through the Body, . . . . .	781
Symptoms of Lead Poisoning, . . . . .	784
Diagnosis of Lead Poisoning, . . . . .	785
Prognosis of Lead Poisoning, . . . . .	785
Treatment of Lead Poisoning, . . . . .	786
 GOITRE— <i>Bronchocele</i> , . . . . .	787
Definition of Goitre, . . . . .	787
Pathology and Morbid Anatomy of Goitre, . . . . .	787
Prevalence of Goitre connected with Limestone Formation, . . . . .	788
Occurrence of Acute Goitre, . . . . .	790
Association of <i>Cretinism</i> with Goitre, . . . . .	790
Definition of <i>Cretinism</i> , . . . . .	791
Three Varieties of <i>Cretinism</i> to be distinguished, . . . . .	791
The Idiocy of <i>Cretinism</i> , . . . . .	791
Virchow's Dissections of <i>Cretins</i> , . . . . .	792
Connection of Goitre with Drinking-Water, . . . . .	798
Nature of <i>Exophthalmic</i> Goitre, . . . . .	794
Treatment of Goitre, . . . . .	794
 PARALYSIS OF THE LOWER LIMBS PRODUCED BY THE USE OF <i>Lathyrus</i> <i>sativus</i> , . . . . .	796
Definition of this form of Paralysis, . . . . .	796
Pathology of the Disease, . . . . .	796
Symptoms and Phenomena, . . . . .	797
Treatment, . . . . .	797

CHAPTER XI.

PATHOLOGY OF THE PARASITIC ORDER OF ZYMOTIC DISEASES, . . . . .	797
Historical Notice and Nature of Parasites, . . . . .	797
List of Genera and Species of Helminthoid Entozoa infesting Man, . . . . .	801
Fecundation, Development, and Migrations of Entozoa, . . . . .	802

CHAPTER XII.

DETAILED DESCRIPTION OF THE PARASITES AND OF THE LESIONS ASSOCI- ATED WITH THEM, . . . . .	805
---	-----

## SECTION I.—THE ENTOZOA.

	PAGE
<b>TAPE-WORMS—Cestoidea,</b>	805
Definition of Tape-worms,	805
Pathology of Tape-worm Parasites,	805
<i>The Mature Tape-worm Parasites,</i>	806
Description of the <i>Tænia solium</i> ,	806
Head, Neck, and Circle of Hooklets of <i>Tænia solium</i> ,	806
Anatomy of the Segments,	807
Growth and Development of a Tape-worm,	808
Segments and Ova of Tape-worms,	809
"Prosclex," "Scolex," and "Strobila,"	810
Stages of Development in a Tape-worm,	811
Description of the <i>Tænia mediocanellata</i> ,	812
Description of the <i>Tænia marginata</i> ,	818
Description of the <i>Tænia elliptica</i> ,	818
Description of the <i>Tænia acanthotrias</i> ,	818
Description of the <i>Tænia nana</i> ,	818
Description of the <i>Tænia flavopunctata</i> ,	818
Description of the <i>Tænia echinococcus</i> ,	818
Description of the <i>Bothriocephalus latus</i> ,	814
Ova of the <i>Bothriocephalus latus</i> ,	815
Description of the <i>Bothriocephalus cordatus</i> ,	815
<i>The Immature Tape-worms, Cystic, Non-sexual, or Vesicular Parasites,</i>	816
<i>Cysticercus cellulosæ</i> ,	816
<i>Cysticercus ex Tænia mediocanellata</i> ,	817
<i>Cysticercus tenuicollis</i> ,	817
<i>Echinococcus hominis</i> ,	817
The Hydatid Tumor or Cyst of the <i>Echinococcus</i> ,	818
Description of the <i>Echinococcus</i> ,	820
<i>Acephalocysts</i> ,	821
Experiments of Siebold and Kuchenmeister,	821
Relation between the Cystic and the Cestoid Entozoa,	821
Summary of Results of the Experiments,	822
How Entozoa gain Access into the Bodies of Animals,	825
Symptoms of the Presence of Tape-worm and Cystic Parasites,	826
Prevention and Treatment of Tape-worm and Hydatid Cysts in Man,	826
<b>FLUKE-LIKE PARASITES—Trematoda,</b>	830
Definition of Fluke-like Parasites,	830
Pathology of Fluke-like Parasites,	830
Development of Distomata,	831
The Distomata found in Man,	833
Lesions in the Urinary Bladder caused by Distomata,	834
The <i>Distoma hæmatobium</i> associated with <i>Hæmaturia</i> at the Cape of Good Hope,	834
Symptoms of the Presence of Distomata,	836
<b>ROUND WORMS—Nematelmia,</b>	836
Definition of Round Worms,	836
Pathological Relations of Round Worms,	836
1. The <i>Ascaris lumbricoides</i> ,	836
2. The <i>Ascaris mystax</i> ,	836
3. The <i>Trichocephalus dispar</i> ,	836
Reproduction of the <i>Ascarides</i> ,	837
Periods of Incubation of the Ova of <i>Ascarides</i> ,	839
4. The <i>Oxyuris vermicularis</i> ,	839
5. The <i>Trichina spiralis</i> ,	840
[Description of the <i>Trichina Spiralis</i> ],	840
Symptoms of Trichinatous Disease,	842
Recent Instances of Trichinatous Disease,	842
Experiments with Trichinatous Flesh,	843
Early Symptoms of Trichinatous Disease,	848
[Trichinous Disease rare in the United States],	849
The <i>Filaria</i> which Inhabit Man,	852
6. The <i>Sclerostoma duodenale</i> ,	853

	PAGE
7. The <i>Strongylus bronchialis</i> , . . . . .	853
8. The <i>Eustrongylus gigas</i> , . . . . .	853
9. The <i>Speroptera hominis</i> , . . . . .	854
10. The <i>Filaria medinensis</i> , <i>Dracunculus</i> , or Guinea Worm, . . . . .	854
Prevalence of the <i>Dracunculus</i> , . . . . .	854
The Number of Worms in one Person, . . . . .	855
Seat or Locality of the <i>Dracunculus</i> , . . . . .	855
Migratory Powers Exhibited by the Guinea Worm before Extraction, . . . . .	856
Anatomical Structure of the <i>Dracunculus</i> , . . . . .	857
Description of the Young, . . . . .	859
Symptoms and Life of the Guinea Worm, . . . . .	860
Period of the Year when <i>Dracunculus</i> is most Prevalent, . . . . .	861
Geological Features of Locality and Soil where the <i>Dracunculus</i> is En- demic, . . . . .	862
Places Infested by the <i>Dracunculus</i> , . . . . .	863
Spontaneous Expulsion of the Guinea Worm, . . . . .	864
Vitality of the Parasite in Water, . . . . .	865
Necessity for the Examination of Water, Mud, and Tanks, . . . . .	865
Nature and Habits of Tank Worms, . . . . .	867
Generation and Propagation of the Guinea Worm, . . . . .	868
Question of the Contagion of the <i>Dracunculus</i> , . . . . .	868
Problems for Solution, . . . . .	869
Other Species of Round Worms, . . . . .	869
<i>Filaria lentis</i> , . . . . .	869
<i>Filaria oculi</i> , . . . . .	870
Treatment of those infested by Round Worms, . . . . .	870
Treatment of Ascarides and Oxyurides, . . . . .	870
ACCIDENTAL PARASITES, . . . . .	871
PENTASTOMA CONSTRICTUM, . . . . .	871
Description of the Parasite and the Lesions it produces, . . . . .	872
Mode of Death caused by the <i>Pentastoma constrictum</i> , . . . . .	875
LARVA or GRUB the Exciting Cause of Bulama Boil, . . . . .	877
Symptoms and Treatment, . . . . .	877
Etiology and Pathology, . . . . .	877
SECTION II.—EPIZOA— <i>Animals living upon the Skin and Hair ; and the Pathology of the Lesions with which they are associated.</i>	
LOUSINESS— <i>Phthiriasis</i> , . . . . .	879
Definition of Lousiness, . . . . .	879
Pathology of Lousiness, . . . . .	879
Various Forms of Lousiness, . . . . .	879
Treatment of Lousiness, . . . . .	882
SCABIES—Syn., ITCH— <i>Scabies</i> , . . . . .	883
Definition of Itch, . . . . .	883
Pathology of Itch, . . . . .	883
Description of the Itch <i>Acarus</i> —Male, Female, and Young, . . . . .	883
Development of <i>Acarus Scabiei</i> , . . . . .	884
Symptoms of Itch, . . . . .	889
The Severe Scabies of Norway, . . . . .	891
Treatment of Scabies, . . . . .	892
SECTION III.—EPIPHYTES— <i>Vegetable Structures ; and the Pa- thology of the Lesions with which they are associated.</i>	
Pathology of Fungus Parasites, . . . . .	894
Vegetable Parasites associated with Disease in Man, . . . . .	897
Pathological Relations of Fungus Parasites, . . . . .	898
Identity of the Fungi described under various Names, . . . . .	901

	PAGE
<b>RINGWORM—Syn., TINEA TONSURANS—<i>Tinea tonsdens</i>,</b> . . . . .	908
Definition of Ringworm, . . . . .	908
Pathology of Ringworm, . . . . .	908
1. Ringworm of the Body, . . . . .	904
2. Ringworm of the Beard, . . . . .	905
3. Ringworm of the Scalp, . . . . .	905
Treatment of Ringworm, . . . . .	906
Epilation, . . . . .	907
<b>FAVUS—Syn., SCALD HEAD—<i>Tinea favosa</i>,</b> . . . . .	908
Definition of Favus, . . . . .	908
Pathology of Favus, . . . . .	908
The Favus Cup and Matter from the Crusts, . . . . .	909
Mycelium, Spores, and Receptacles, . . . . .	909
Symptoms of Favus, . . . . .	910
Varieties of Scald Head, . . . . .	912
Treatment of Favus, . . . . .	918
<b>BALDNESS IN PATCHES—Syn., TINEA DECALVANS—<i>Tinea decalvans</i>,</b> . . . . .	914
Definition of Baldness, . . . . .	914
Pathology of Baldness, . . . . .	914
Nature of Vitiligo, . . . . .	915
Treatment of Baldness, . . . . .	916
<b>TINEA [PITYRIASIS] VERSICOLOR, LIVER-COLORED SPOTS, OR CHLOASMA,</b> . . . . .	916
Definition of Chloasma, . . . . .	916
Pathology of Chloasma, . . . . .	916
Treatment of Chloasma, . . . . .	917
<b>MYCETOMA—Syn., MADURA FOOT—<i>Mycetoma</i>,</b> . . . . .	918
Definition of Mycetoma, . . . . .	918
Pathology and Historical Notice of Mycetoma, . . . . .	918
Varieties of Mycetoma, . . . . .	919
Fundamental Cell-Structure of Mycetoma, . . . . .	928
[Description of the Varieties of Fungi in Mycetoma], . . . . .	923
Symptoms of Mycetoma, . . . . .	925
Description of Incipient Mycetoma, . . . . .	926

LIST OF ILLUSTRATIONS IN VOL. I.

Diagram of the Typical Range of Temperature in Cases of <i>Natural Small-Pox</i> , .	238
Diagram of the Typical Range of Temperature in cases of <i>Small-Pox modified by Vaccination</i> , . . . . .	247
Diagram of the Typical Range of Temperature in Cases of <i>Measles</i> , . . . . .	289
Diagram of the Typical Range of Temperature in Cases of <i>Scarlatina</i> , . . . . .	302
Diagram of the Typical Range of Temperature in Cases of <i>Erysipelas</i> , . . . . .	328
Diagram of the Typical Ranges of Temperature Contrasted in Cases of <i>Typhus</i> and <i>Typhoid Fever</i> , . . . . .	357
Diagram of the Typical Range of Temperature in Cases of <i>Typhus Fever</i> , . . . . .	399
Diagram of the Typical Range of Temperature in Cases of <i>Febricula</i> , . . . . .	482
Diagram of the Typical Range of Temperature in Cases of <i>Protracted Febricula</i> , . . . . .	488
Diagram of the Typical Range of Temperature in Cases of <i>Quotidian Ague</i> , . . . . .	478
Diagram of the Typical Range of Temperature in Cases of <i>Tertian Ague</i> , . . . . .	474
fig.	
A and B, Clinical Thermometers for Physicians, . . . . .	59
[C, D, E, Sections of Ulcerated Colon in Chronic Camp Diarrhœa], . . . . .	569
1. Condition of Heart, Lungs, and Great Vessels after Death during Collapse of Cholera (JOHNSON), . . . . .	578
2. Fungi and their Development by Cultivation from the Rice-water-like Stools of Malignant Cholera (ERNEST HALLIER), <i>to face</i> . . . . .	591
3. Changes in the Blood-Corpuscles, subsequent to the Bite of the <i>Cobra di Capella</i> (HALFORD), . . . . .	648





# THE SCIENCE AND PRACTICE OF MEDICINE.

---

## PART I. TOPICS RELATIVE TO PATHOLOGY.

---

### CHAPTER I. OF MEDICINE AS A SCIENCE AND AS AN ART; ITS OBJECTS AND ITS EXTENT.

THE study of MEDICINE is prosecuted under two relations, namely, as a *Science* and as an *Art*. MEDICINE, considered as a *Science*, takes cognizance of all that relates to our knowledge of diseases; and, especially, of the circumstances under which they become developed, of the conditions of their existence, of their nature and of their causes in the widest sense of these terms. Considered as an *Art* (in so far as Medicine has that practical value), its object is to distinguish, to prevent, and to cure diseases.

The object and aim of MEDICINE as an *Art* is to alleviate human suffering, and to lengthen out human existence, by warding off or by modifying disease "as the greatest of mortal evils," and by restoring health, and even at times reason itself, "as the greatest of mortal blessings." In other words, the practical view required to be taken of MEDICINE is, that "it is the art of understanding the nature of diseases, so as to appreciate their causes, and to prevent their occurrence when possible; to promote their cure, or to relieve them when they occur." (BIGLOW.)

Many branches of human knowledge are combined in the constitution and elucidation of the *Science*; and the practice of Medicine as an *Art* ought to be founded on principles and facts of universal, or at least of extensive applicability.

A consideration of the different topics which together make up the Science of Medicine suggests a division of the subject into the following departments, namely: (1.) PHYSIOLOGY, which embraces the study of the healthy functions of which the human body is the

seat or instrument ; (2.) PATHOLOGY, subdivided into *Special Pathology* and *General Pathology*, which together embrace a consideration of everything relative to the existence and nature of diseases ; (3.) THERAPEUTICS, which expounds the various actions of remedies upon the diseased economy, or the means by which nature may be aided in her return to health ; (4.) HYGIÈNE, which embraces a consideration of the means of preventing disease, or, in other words, of preserving health.

Physiology, General Pathology, Therapeutics, and Hygiène, are sometimes designated indifferently by the titles of the "*Institutes*," the "*Institutions*," or the "*Theory of Medicine*."

These departments of science are all preliminary subjects of study, and constitute a necessary and appropriate introduction to the Practice of Physic, in which Special Pathology and the treatment of special diseases are the leading topics of consideration.

Each of these departments has grown or expanded itself into a great branch of science ; and any single section is sufficient of itself to occupy the lifetime of an individual in working out and studying it in detail. It is therefore not possible for the human mind to embrace all of these departments in their whole extent or relations to each other ; and, setting aside the consideration of theories and systems, it has been truly observed, "that no man possesses all the pathological knowledge contained in the records of his art" (CHOMEL) ; and it is, therefore, far less possible to embrace in any single treatise a view of the Science of Medicine in all of these departments.

For the purpose of teaching the Science of Medicine in its application to practice, its elementary principles, as developed in the departments of Pathology, are the most useful guides to the student.

---

## CHAPTER II.

### HOW THE PROVINCE OF PATHOLOGY IS MAPPED OUT.

AN inquiry into the nature of diseases embraces a consideration of the following topics : (1.) *The accurate observation and correct registration of facts in Pathology.* On the efficiency of the machinery devised for these important ends will rest our power, to curb the invasion of our science by the guesswork of theory. Such records must be the means eventually of rooting out the traditional errors which so largely pervade medical literature. (2.) *Descriptive Pathology, embracing GENERAL and SPECIAL Pathology.* Special Pathology is intended to comprehend a consideration of the essential nature and origin of particular diseases as they occur in man and animals, and General Pathology to include those more general facts or principles which result from a comparison of particular diseases with each other. Although Special Pathology comes first in the order

of Nature, yet, wherever the arrangements for Medical education are complete, General Pathology is taught as an introduction to, or conjointly with, the special study of diseases, just as in other sciences—for example, in chemistry—it is found convenient to give a general view of the principles which have been established by experiment and observation, before entering upon the particular details of the science. All theory in Medicine; all descriptive Pathology; all grounds for rational speculation regarding the nature of diseases, and for the framing of experiments; as well as all maxims of practice which aim at the prevention or cure of diseases, must rest ultimately on observed and recorded facts. *Accuracy of observation* is therefore the first lesson the student has to learn in all methods of investigation, and the lesson is one of paramount importance. The best observations, however, will avail but little unless the observed facts are recorded in such a way as to secure their preservation; and descriptive pathology mainly concerns itself, in the first instance, with the *Registration of facts*, as embraced,—

- (1.) In the *History of cases of disease* from their origin to their end.
- (2.) In the *Statistics of disease*. Such registration includes methods for preserving, in an authentic and permanent form, the memory of facts in Pathology as they occur. It thus eventually furnishes materials upon which future Pathologists and Statists will build a comprehensive and definite system of scientific Medicine. It will furnish the means of teaching all that is necessarily involved in our notions regarding the nature of diseases. The descriptive Pathology so arrived at considers diseases as they exist, or have existed in man, in the lower animals, or in plants. It considers the conditions under which diseases originate; it considers how far certain conditions are fulfilled before disease establishes itself; and it aims at demonstrating how far such conditions are inconsistent or incompatible with the maintenance of health. Subsequently, with extended information, descriptive Pathology may undertake to assign the conditions which give rise to certain diseases rather than to others. It will eventually define the elements necessary to establish, to originate, or to constitute particular diseases; and will show how the same disease, or class of diseases, may assume various forms, but in all of which definite elements are recognizable. Descriptive Pathology thus aims at determining and describing the essential elements of a disease.
- (3.) *Speculative Pathology* assumes that we know what a disease is—that we know the effects it produces—that we know the conditions necessary for its existence—that we know its relations to other diseases. It seeks to inquire how certain conditions or circumstances will operate in bringing about disease. It seeks to determine the tendency in the future of a diseased state from certain observed facts in its course, or in the course of similar diseases. Statistical data are thus the main basis of its operation.
- (4.) *Pathology dictates the maxims of rational practice*. It is in the nature of the science of Pathology that it always ought to be in advance of our certain knowledge regarding the treatment of diseases. It is the basis of rational medicine; for it is rational to know the nature of a disease, in order (1.) To enable us to prevent

it; (2.) To understand the principles which ought to guide us in the management of it. Such are the main divisions which the province of Pathology embraces.

It is intended, however, in the first part of this handbook merely to guide the student to notice,—(1.) The *relative* nature of the terms “*Life*,” “*Health*,” “*Disease* ;” (2.) How the nature of diseases may be elucidated ; (3.) The nature of the morbid phenomena, symptoms, and signs of disease ; (4.) The means and instruments of investigation into the nature and causes of disease ; (5.) Some of the more elementary constituents of disease ; (6.) Some complex morbid states associated with individual diseases, or with conditions of ill-health (*cachexiæ*) ; (7.) The modes by which diseases terminate fatally ; the types of disease and their tendency to change ; (8.) The general treatment of the complex morbid processes.

In the three subsequent parts of this work it is intended to consider some of the details of the science and practice of Medicine, to furnish the student with,—(1.) A nosological system by which to classify and name diseases. (2.) A detailed description of characteristic diseases in the respective classes of that nosological arrangement. In this part a definition (provisional) and a history of the nature of each disease will be given ; the probable course and succession of events will be described, and the grounds on which an accurate diagnosis may be made, or a prognosis expected ; and, lastly, a detailed account of those rational modes of treatment which are consistent with the established principles of the *Institutes of Medicine*. (3.) An account of what is known relative to the geographical distribution of diseases.

---

## CHAPTER III.

### RELATIVE NATURE OF THE TERMS LIFE, HEALTH, DISEASE.

THE word *Disease* is used in a general and also in a specific sense ; as when it is said that a person is diseased, without the nature of the affection being stated ; or, that he suffers from a particular disease, such as small-pox. Attempts to give a precise definition of the term *Disease* have all been unsuccessful. The relations of the morbid state to the condition of health, and of health to the performance of the vital functions, are of such a kind that they can merely be described in connection with each other, but not defined.

If *life* is understood to imply an active state, resulting from the concurrent exercise of the functions of the body, then there are conditions of activity and of mutual adaptability of functions and of parts, both as regards body and mind, which are necessary to healthy existence. Our notions of *the conditions of health* have thus considerable latitude. *Health* is merely a name we give to that

state or condition in which a person exists fully able, without suffering, to perform all the duties of life. Many degrees of this state are therefore at first sight obvious, from the possession of a feeble existence to the most robust condition of the body; and there are many degrees of feebleness and delicacy of health which we cannot say are due either to disordered or diseased states of the frame. Our notions of *normal life* are thus so extremely indefinite that it is only by a forced abstraction the normal can be separated from the abnormal. Hence also our idea of *disease* is very indefinite; it cannot be separated by any well-defined boundary from our idea of *normal life*, and the two conditions are connected by a kind of debatable border land.

When we regard, therefore, the phenomena of the living state and the conditions of health, we can readily observe when and how *disease* is but A DEVIATION FROM THE STATE OF HEALTH, CONSISTING FOR THE MOST PART IN A CHANGE IN THE PROPERTIES OR STRUCTURE OF ANY TISSUE OR ORGAN, WHICH RENDERS SUCH TISSUE OR ORGAN UNFIT FOR THE PERFORMANCE OF ITS ACTIONS OR FUNCTIONS ACCORDING TO THE LAWS OF THE HEALTHY FRAME.

It is now a received pathological doctrine that *disease* does not consist in any single state or special existence, but is the natural expression of a combination of phenomena, arising out of impaired function or altered structure. All attempts, therefore, to define *disease* by the use of such terms as "*derangement*," "*modification*," "*alteration*," "*change*" from the pre-existent state of health, show, in the first instance, that very various ideas are attached to the term or to the state; and, secondly, that these terms point to a nosological division into structural and functional disease, rather than to a state common to all forms of disease.

A definition of any state of disease ought to include all the circumstances, whether functional or organic, which constitute the deviation from health; and for very obvious reasons such a definition can only be approximately expressed.

---

## CHAPTER IV.

### HOW THE NATURE AND CAUSES OF DISEASES MAY BE ELUCIDATED.

THE nature of the derangements to which the human body is liable may be studied under the three following aspects: (1.) As diseases present themselves in individual cases, becoming thereby the subjects of CLINICAL INVESTIGATION and INSTRUCTION—a method of teaching in which the *Natural History* of the disease ought to be a special subject of study; (2.) As they constitute particular genera or species of disease, forming the topics of SPECIAL PATHOLOGY; (3.) As they may be reduced to and studied in their primary elements, forming thereby the science of GENERAL PATHOLOGY.





exists. (2.) How far the condition of the patient is removed from the state of health usual to him. (3.) As to the nature of the disease, and how it is distinguished from other ailments, or in what respects it may differ from the same ailment in other people in similar circumstances. Thus a *diagnosis* is made by the art of converting *symptoms* into *signs* of disease.

But the physician at the same time generally carries his mental exertion a little further. He tries to arrive at a just estimate of the probable result or *event* of the malady, and so makes up his mind,—(4.) As to whether the illness will terminate in the death of the patient, in permanent organic mischief of greater or less extent, in persistent impairment of the general health (*cachexiæ*), or in complete recovery. As in Politics, so in the science of Medicine: the Politician and the Physician have each to deal with the future, as well as with the present. Both endeavor to *forecast* events; and thus, in the practice of Medicine, we are said to make or give a *prognosis*. (5.) The Physician must be able also to appreciate with reasonable rapidity those *symptoms* which are peculiar, and to recognize them when associated together as the *signs* of particular or definite morbid states. Such *symptoms* are then said to furnish *pathognomonic signs* of disease. (6.) The Physician must further discriminate, and try to put a fair and just value or interpretation upon, those symptoms which are only experienced by the sensations (subjective) of the patient himself, as contrasted with those which may be seen or appreciated by others—such as objective phenomena or physical signs.

The interpretation of symptoms can only be successful after a close observation of the patient—often prolonged, and repeated for more complete investigation—so as to connect the results arrived at with his previous history. The utmost logical acumen is required for the due interpretation of symptoms. The individual value of each ought to be duly weighed; one symptom must be compared with another, and each with all; while the liability to variation of a similar symptom in different cases of a like kind must not be forgotten; and the occasional absence of the usual pathognomonic signs may be sometimes calculated upon. Thus only can the nature of a disease be clearly determined—its severity and dangers fully appreciated—its treatment indicated, and the probability of recovery foretold.

A close observation of general symptoms, in all their details, is absolutely necessary; and the investigation is aided practically by the improved instruments of the present day, and better methods of examination. Above all things, *methodical examination* is essential for the student, if he would acquire the habit of carefully and accurately examining the nature of the cases of disease with which he will have to deal. Patients must be examined methodically, in order that the symptoms of disease may be correctly interpreted, and that nothing be overlooked or neglected. Directions have been given by many authorities for acquiring and habitually following a definite system of examining patients, as to what are the essential data to be obtained and recorded in case-taking; and





origin. It is but yet in process of development, although its foundations may be traced in the works of the earliest medical writers of antiquity. All of them refer to changes which they *merely supposed* had taken place in the internal organs; and they were doubtless led to this assumption by observing the connection that existed between structural lesions of the external parts and their accompanying symptoms. Hippocrates describes the deposit of tubercles in the lungs, the symptoms occasioned by them in a crude state, and those which attend their softening and discharge.

The science of MORBID ANATOMY is a record of facts. In its relation to the progress of Medicine it is a living record—a history whose pages must be ever open to receive the observations which are constantly being made by those engaged in pathological pursuits—a record from which one may ascertain at any time the conditions under which morbid changes or new formations in the body have taken place. The pages of this history show that at the present day the department of pathology is in a transition state; and the position of Medicine, as a science, must eventually result from a re-arrangement of the innumerable details which the sciences of morbid anatomy and histology may disclose and unfold. It is necessary, therefore, and often advantageous, to look back upon the past, and see what has already been done, so that its venerable facts may not be lost sight of, but grouped in series with the extensively verified experiments and observations of the present day. In so doing, if we pause and contemplate the steps which have been taken to arrive at our present position, such a contemplation may stimulate the youthful student to the noblest exertions of his intellect, as he can not fail, with extensive study, to see before him, and on every side, much unlabored but productive soil. Such a retrospect will at the same time have the effect of placing in a prominent aspect the varied influences which morbid anatomy has had on the science of Medicine, the conditions under which it has flourished, and the legitimate objects of its investigations.

The art of printing had not been long invented when books on morbid anatomy began to issue from the press; and although, the early period of the fifteenth century has left little enduring literature of any kind (but has been mainly distinguished by the number of colleges then founded), yet about this time pathological anatomy in the medical school of Florence shows the earliest evidences of an existence.

The facilities for study which the art of printing introduced soon stirred up ardent students; and the sixteenth and seventeenth centuries produced much that will ever remain famous in the annals of medical science. Eustachius, Tulpus, Ruysch, Harvey, Malpighii, and Leuwenhoeck are names familiar as household words to the student of medicine. The earlier attempts of this period to form a system of pathological anatomy is characterized by abortive endeavors to explain all results upon some exclusive and general principle. A spirit of speculation marks the character of the age. The men of that time had observed but few facts; and on these facts they preferred to speculate and dogmatize, rather than prosecute the



Forbes, who but recently has taken his place amongst the "Great ones of the Past," emphatically recorded the observation more than fifteen years ago, that "here the surest and most glorious triumphs of medical science are achieving, and are to be achieved." He himself lived to see great and good results; to see improvements in social and sanitary matters which continue to be realized, and whose rapid progress is characteristic of the present period. Within the last half-century land-draining and town-sewering have ripened into sciences. From rude beginnings, insignificant in extent, and often injurious in the first instance, the systematic sewerage of towns and draining of land have become of the first importance. Land has thus, in not a few instances, doubled its value. Town-sewering, with other social regulations, have contributed to prolong human life from 5 to 50 per cent. as compared with previous rates in the same district. Agues and typhoid fevers are reduced in the frequency of their occurrence. Since 1840 an annual mortality in English towns of 44 in 1000 has been reduced to 27; an annual mortality of 30 has been reduced to 20, and even as low as 15. Not less remarkable reductions have taken place in the mortality and loss of strength in the army and navy; so that generally it may be said that human life has now more value in England than in any other country in the world—a result entirely due to better sanitary arrangements (Rawlinson "On Sewering of Towns," *Soc. of Art Journal*, vol. x, p. 276).

The political economist can not now, therefore, regard MEDICINE in any other light than as a productive art; and the labors of the Physician, whether in civil or in military life, can not be regarded as unproductive labor.

But the science of Physiology (on which much of our sanitary improvements are based) has immeasurably outstripped the science of Pathology in the comprehensiveness of its views and in the value of its results; while Pathology, in its turn again, has always been, and ought to be, in advance of Therapeutics. The best physiologists have distinctly recognized that the basis of the science must include not only a knowledge of animals below man, but a knowledge of the entire vegetable kingdom. Without such an extensive survey of the whole realm of organic nature, we cannot possibly understand human physiology, and far less comparative physiology. The science of Pathology, therefore (whose aim is to expound the *nature of diseases*), must be, *à fortiori*, very far behind. The diseases of the lower animals, for instance, rarely form any part of the study of the student of Medicine. The diseases of plants are almost entirely neglected. Yet it is clear that until all these have been studied, and some steps taken to generalize these results, every conclusion in pathology regarding the nature of diseases must be the result of a limited experience from a limited field of observation. How do we know that the blights of plants, or the causes of them, are not communicable to animals and to man? We know how intimately related the diseases of man and animals are with famines and unwholesome food; and of famines with the diseases of vege-



the textures, organs, and functions of the body whose normal exercise constitutes a healthy existence (LONGET, MULLER, SHARPEY, VALENTIN, ALLEN THOMSON, CARPENTER, KIRKES, PAGET, KÖLLIKER). (3.) By an intimate knowledge of the normal development of the human textures, as well as those of plants and animals from the fecundated ovum (BISCHOFF, COSTA, ALLEN THOMSON, HUXLEY, NEWPORT, and KÖLLIKER). (4.) Besides these kinds of investigations, the science of practical medicine has been, and is being, advanced by operations and experiments upon the internal organs of living animals, opprobriously termed *vivisections*. At some of our great schools of medicine such investigations are now being actively but judiciously prosecuted and taught; as by Bernard, in Paris, Drs. G. Harley, Brown-Séquard, and Pavy, in London.

Successful inquiries into Pathology, or the nature of diseases, cannot be said to have commenced till the middle of the eighteenth century, when the great work of Morgagni issued from the press. It was the work of his lifetime. In the eightieth year of his age, and not till then, did he consider himself warranted to publish his observations, *De Sedibus et Causis Morborum* (1761); a work whose material and circumstances of publication read us the practical lesson, that the more frequently a disease occurs, the more necessary it is that its phenomena should be carefully investigated. And when we think of the prudent reserve, the anxious and the conscientious delay exhibited by Harvey, Morgagni, and Jenner, in the publication of their respective researches, we cannot but contrast the circumstances with those under which the exuberance of medical publications are now given to the world. Morgagni modified and corrected many of the views entertained and promulgated by his predecessors; and the study of the nature of diseases was carried into the commencement of the present century by CULLEN, DE HÄRN, WILLIAM and JOHN HUNTER, PORTAL, and BICHAT.

The knowledge of the physician regarding the nature of disease-processes may now be observed to have advanced simultaneously with that of *general anatomy*; and when the component parts of an organ, and of the human body, came to be distinguished, it was soon observed that membranes and tissues might be individually diseased while neighboring membranes and tissues remained untouched. Bichat's idea, therefore, of decomposing the animal body into its elementary parts, must be regarded as the foundation of modern special pathology; and while he pointed out the necessity of studying diseases with reference to the different tissues as separately and specially affected, it has been since shown, in a remarkable manner, how general anatomy, deduced from physical properties of parts and crude observation, may coincide with more minute investigations of a chemical and microscopical kind. The membranes and tissues composing the organs of the body, roughly torn asunder by Bichat, are now themselves being daily subjected to a more inquisitive analysis of an anatomical and chemical nature, which unravels them into still more minute histological elements.

Although, therefore, Bichat entertained the view that each tissue had its own *diathesis*, it is to Cullen and the Hunters in this country



days desired to know nothing but anatomy and mechanics; and, accordingly, it may be recognized as the period of pure anatomical and mechanical surgery, distinguished by the writings of men whose works bear ample testimony that the surgery of the period was founded on exact and even minute anatomical knowledge. No allusion is made, however, by them to medicine,—they make no application of physiological truths, and they encourage no therapeutic tendency apart from mechanical or instrumental interference.

The purely solidist, as well as the purely humoral principles by which the nature of diseases have been explained, may be said to have died a natural death long ago; but, as already noticed, the remembrance of what is valuable in the results of both are preserved in modern pathology, which takes its stand upon anatomical and physiological facts, connected by simple methods of inductive observation with the symptoms and signs of disease as seen and expounded to the student by the distinguished professors of Clinical Medicine at most of our celebrated schools, where Clinical Medicine is taught.

In this field of instruction it seems invidious to mention here the names of men still living. For their own sakes, as well as for science, may they be long deprived of being thus honorably and respectfully mentioned. As teachers, they are in our own country familiar to every student. As recorders of what they observe at the bedside and after death, they are not less celebrated abroad than appreciated at home.

Tested by extensive clinical observations, the character of the present period in the history of Practical Medicine is one of *probation* as well as of *progress*, marked by a close inductive examination of past generalization and classification of facts, however remotely connected, which illustrate the nature of diseases and their treatment.

Side by side, since 1816 and 1819, the microscope and the stethoscope, under the influence of such men, have advanced our knowledge of the nature of diseases with a regular and accelerated velocity; but they have only done so as assistants and in subordination to laws and facts whose knowledge we have acquired by a close observation of general symptoms, of which such instruments have never been intended to take precedence. They have never accomplished, nor can they ever accomplish, useful practical results, to the exclusion of such other methods of observation as have just been noticed. We are not to confound *relative* smallness with *absolute* simplicity, and believe that because a simple organic cell is a small object—because we can see around it, through it, and on every side of it—the functions and conditions of its existence are less *complex* or less *obscure* on that account than are those of a more complex organ, or the functions of a living body.

We are not to suppose that because the stethoscope enables us to detect a mitral murmur, or a crepitation in a lung, we are justified at once in adopting one, and only one, method of treatment. It is this exclusive use of instruments, to the disregard of general





thermometrical control, he may soon fall into gross errors. So it is with the ophthalmoscope, specula, and all other more or less exact physical aids to diagnosis. Let them be in constant and appropriate use, but the results must always be taken and compared in connection with other general symptoms of disease.

In all the temperate regions of the world, histology, as applied to morbid products, has been cultivated, and has advanced our knowledge regarding disease ever since 1838. In warmer latitudes our knowledge of practical Medicine has been advanced by extensive observations on physical climate, medical topography, and by organic chemical analysis applied to obtain therapeutic agents from the vegetable world. Those may be said to be the characteristics of the researches of our own country, Germany, France, and America, as contrasted with the nature of the observations mostly prosecuted in India.

No exclusive doctrine will now stand the test of well-directed pathological inquiry, the main object of which is to connect all organic changes (lesions) and functional derangements with their symptoms and causes, with the view of applying rational remedies and prophylactics. The too exclusive study of pure organic pathology and morbid anatomy leads to no distinction between the signs and causes of disease; and the obvious tendency of such exclusive study is to exaggerate the importance of the principles it may establish, to hold out no hopes of cure, and to undervalue the power of remedies and remedial measures. To obviate this tendency it is necessary to have recourse to inductive reasoning, so as to connect all the morbid changes seen or appreciated after death with the signs and symptoms of disease observed during life. Thus it is that links in the chain of disease-processes which, from a one-sided or exclusive view, appear isolated and localized, are really found to be connected with each other. It may be, also, that they are connected with a long but intelligible series of processes developed during life through the metamorphosis of tissue, and going on in apparent health, or in an obviously morbid exercise of function. The constitutional origin of many local diseases, otherwise inexplicable, then becomes apparent.

Among the more eminent exponents of this rational school of pathology, who at an early period in this country discerned and appreciated such doctrines, we find the names of ALLEN, GOLDING BIRD, SIR ROBERT CARSWELL, GREGORY, HOPE, HODGKIN, MARSHALL HALL, PROUT, WILLIAM STARK, JOHN THOMSON, TWEEDY TODD, and many others, who, although now no more, have left behind them imperishable evidence of their labors. The younger pathologists of the present day, whose name is *Legion*, follow in the footsteps of these men, extending the fields of observation and the boundaries of the science of Medicine. By them the importance of morbid anatomy is sufficiently appreciated, and its province distinctly defined and limited as follows, namely: (1.) To detect the changes which have taken place during the course of diseases in the structure of tissues and organs of the body; (2.) To demonstrate the

exact seat of local alterations established during the progress of disease.

The investigation and elucidation of the *nature, course, and causes* of those changes, constitute the prominent objects of the science of pathology. By the aid of morbid anatomy and clinical observation during life, pathology seeks to establish the relations of the changes which lead to the lesions, and so to connect the general progress of disease with its symptoms and signs.

MORBID ANATOMY goes beyond its province when it attempts to point out the nature of the proximate cause of disease. It is only by the application of inductive reasoning that the connections of causes and morbid effects can be shown, and such constitutes the main object, and is the highest aim of the science of PATHOLOGY.

The *morbid anatomist* finds a lesion or change for what ought to be the natural structure, appearance, or condition of a part. The *pathologist* seeks to connect such lesions with signs and symptoms during life, that the *practical physician* may suggest a remedy to the disease, and that the *nosologist* may give it a name, distinguishing characters, and a place in his classification.

---

## CHAPTER VII.

### THE ELEMENTARY CONSTITUENTS OF LESIONS AS SHOWN BY MORBID ANATOMY AND OTHER MEANS OF RESEARCH.

WHERE the material effects of disease can be rendered obvious they are found to consist for the most part of,—

1. Morphological changes in the elementary textures of the body generally, and altered conditions of the fluids.
2. The presence of new formations foreign to the normal condition of an organ or system of organs.
3. Change in the position or form of some of the organs or parts of organs.
4. Deposits in or around the elementary parts of tissues, or changes of a degenerative or retrograde kind in them.

The object of prosecuting the anatomy of disease is, therefore, in the first instance, to institute a comparison between the known appearances or standard of health and an altered state of the parts. Such a comparison is, in the first instance, founded on an intimate knowledge of the doctrines stated at page 36.

**Means and Instruments of Research.**—To institute investigations such as those indicated at page 40, advantage must be taken of almost every branch of human knowledge. The methods of carrying on pathological research are therefore very varied, but may be shortly enumerated under the following heads:

1. The opening of dead bodies, to ascertain the condition of their

organs and tissues in all that relates to their structural, chemical, and physical properties (ROKITANSKY, HASSE, VIRCHOW).

2. Application of various instruments, such as the microscope, and of means to ascertain the absolute and specific weight of organs or parts, the relations, size, form, and colors of structures, and the like (QUEKETT, BENNETT, BEALE, PEACOCK, BOYD).

3. Application of chemical investigations to the diseased products (VOGEL, SIMON, DAY, LEBERT, GLUGE, BEALE, GARROD, CHRISTISON, PARKES, VIRCHOW, FRERICHS, GAIRDNER).

4. Application of statistics to determine various points of interest in reference to the nature, course, and complications of diseases (WM. FARR, GUY).

5. Means to preserve objects for further study by the microscope, or any other mode of examination (TULK, HENFREY, BEALE, QUEKETT, VAN DER KOLK, LOCKHART CLARKE).

6. Experiments instituted on living animals, and, in certain cases, on man, with the view of artificially producing a morbid condition. A careful study of such experiments by the previously mentioned means affords valuable information, for the causes in action are more under control than those which are spontaneously brought about by disease in the living body (BERNARD, HARLEY, PAVY, KUCHENMEISTER, ZENKER, and others).

The immediate object of such investigations is to obtain information regarding the material changes in the different parts of the body which accompany or produce morbid symptoms, and to connect these changes with symptoms and signs of disease during life. We thus learn how morbid products are formed at first and gradually perfected; and by combining these two kinds of knowledge we learn the relative connection of two orders of phenomena; namely, how the perverted properties, disordered actions, or altered structures give rise to perverted or impaired secretions; disordered and irregular motions; deranged, impeded, or interrupted functions. In other words, the "*order of invasion of disease-processes*" is learned from such investigations; and we are thereby taught how parts, once the seat of morbid change, return, by various processes of nutrition, growth, repair, or reproduction, to their normal condition.

The questions arising out of such investigations are, or ought to be, the first object of thought to the conscientious medical practitioner. It is his duty, from an attentive consideration of the signs and symptoms of disease, to form an idea, as accurate as possible, of the nature and extent of the morbid action or change which is going on, or which may be set up, in the tissues, organs, and fluids of the living body.

If, therefore, he does not avail himself of every means and instrument by which he can ascertain the existence of change in the dead body, and its alteration from some standard of health—if he does not embrace every opportunity of making post-mortem examinations—if he contents himself merely with observing signs or symptoms of disease, without witnessing the changes of structure, if any,

which may give rise to them—he can have little conscious satisfaction in the study of Medicine as a science, or in the practice of the healing art. In the words of Cruveilhier, he will, during his lifetime, “see many patients, but few diseases.” Such a practitioner is not to be trusted.

### *Various Forms of the Constituent Elements of Disease.*

The histologist has now clearly ascertained the various simple organic forms which compose the textures in their normal state, and the mode in which these textures are arranged and combined so as to form the organs and systems which carry on the healthy functions of the body. The pathologist has made out (although with less completeness), by the methods of observation and experiment already indicated, the various simple organic forms which constitute the elements of those material changes whose phenomena of growth, decay, and varied change are associated with the manifestations of disease. By classifying and arranging these forms we obtain more clear ideas of lesions; and we ascertain that the material morbid processes follow, in their development, a very definite order of change, not yet in all cases determined with absolute certainty.

An anatomical investigation of morbid parts, conducted with the aid of the microscope and other instruments of research, shows that the material of which their substance is made up is of very various structure, sometimes combined in forms of one kind throughout, and sometimes varied by the development and combination of many elementary forms, more or less solid, soft, or fluid.

An analysis of the morbid material, carried as far as scientific means at present enable us, shows that the elementary conditions in which morbid products are found may be described as follows:

1. Fluid matter and hyaline substance, more or less soft.
2. Simple elementary forms of the nature of deposits, sometimes of a mineral or inorganic character; *e. g.*, (*a.*) amorphous granules; (*b.*) crystalline structures in a granular state.
3. Simple, but organized products capable of growth; *e. g.*, (*a.*) granules; (*b.*) compound corpuscles; (*c.*) simple cells; (*d.*) fibres.

The various appearances and conditions which these simple forms may assume in disease, as well as the functional states with which they are frequently associated, lead to a further enumeration and classification of morbid elementary products, as well as of more complex disease-processes, as below:

### **A.—MORBID ELEMENTARY PRODUCTS.**

#### **I. EXUDATIONS MORE OR LESS SOFT, SEMI-FLUID, OR FLUID, AND FORMED OF,—**

*a.* Germinal plastic and formed material, which has sometimes been called *blastema*, *coagulable lymph*, *false membrane*, or *fibrine*, as seen adhering to free surfaces.

*b.* Aqueous matter, as seen in the morbid state termed "*dropsy*," and "*œdema*" of parts.

*c.* Gaseous exudations, as seen in the various forms of *pneumato*sis.

## II. EXUDATIONS MORE OR LESS CONSOLIDATED, AND CONSISTING OF,—

*a.* Molecular or granular material, from the 800th of a line to an immeasurably small size, and consisting chiefly of the simple forms of,—

(1.) Fatty molecules or granules.

(2.) Forms of an organic kind capable of growth, and invariably taking origin from a pre-existing structure.

(3.) Deposits of an inorganic kind, generally calcareous salts.

(4.) Pigment granules.

*b.* Coagulable compounds, resisting the action of most reagents, such as are seen in the elements of tubercle, scrofula, oleo-albuminous formations.

*c.* Exudations of a transitional nature, organized, which are capable of growth, which may become vascular, which grow from pre-existing structures, and which are composed of,—

(1.) Consolidated homogeneous material passing to

(2.) A fibrilloid arrangement of the molecular or granular particles composing connective substance, and a subsequent formation of fibres in it or from it.

(3.) The formation of pyoid cells, and fibro-plastic or connective tissue cells, passing into fusiform cells and fibres as the material becomes consolidated.

(4.) The formation or exudation of fluid matter holding pus, or other more compound cells.

## III. GROWTHS AND EXUDATIONS OF A MORE OR LESS SPECIFIC KIND.

*a.* Lymph of small-pox and cow-pox.

*b.* Matter of glanders, of malignant pustule, and of the plague.

*c.* Fluid of infecting chancre, and of some forms of secondary syphilitic lesions.

*d.* Material of tubercle and scrofula. (?)

*e.* Material of cancer.

*f.* The growth in Peyer's glands during typhoid fever.

*g.* The growth in Peyer's glands in cases of cholera.

*h.* Melanotic or pigmentary germs.

## IV. MATERIAL OF A COMPLEX KIND.

*a.* Media of repair and reproduction of injured or lost parts—substance of granulations and cicatrices.

*b.* Hypertrophy of parts.

*c.* Tumors,  $\left\{ \begin{array}{l} \text{innocent.} \\ \text{malignant.} \end{array} \right.$

*d.* Concretions.

## V. PARASITIC FORMATIONS.

**B.—COMPLEX VITAL PROCESSES WHOSE PHENOMENA, MORE OR LESS COMBINED, CONSTITUTE DISEASE.**

1. *Fever*—the febrile state—*Pyrexia*.
2. *Inflammation*.
3. *Irritation*.
4. *Congestion*.
5. *Depression*.
6. *Atrophy*.
7. *Degeneration*.

Such a classification as the above is merely intended to bring before the student at a glance the variety of morbid material which is concerned in the expression of many of those phenomena seen in the course of disease, the distinctions made being mainly based on structural analysis.

While it is more properly the province of the anatomist to describe the MORBID ELEMENTARY PRODUCTS, it is the COMPLEX VITAL PROCESSES, WHOSE PHENOMENA, MORE OR LESS COMBINED, CONSTITUTE DISEASE, with which the Physician has more immediately to deal; and some of these complex states especially require notice here; namely, *Fever and Inflammation* and some forms of *Degeneration*.

---

## CHAPTER VIII.

### COMPLEX MORBID STATES.

#### SECTION I.—FEVER—*Pyrexia*.

**Definition.**—*A complex morbid state which accompanies many diseases as part of their phenomena, more or less constantly and regularly, but variously modified by the specific nature of the disease which it accompanies. It essentially consists in elevation of temperature, which must arise from an increased tissue-change and have its immediate cause in alterations of the nervous system (VIRCHOW, PARKES).*

**Pathology of Fever and Phenomena which constitute the Febrile State.**—In describing the nature of fever, the following statements are principally compiled from the Gulstonian Lectures of Dr. Parkes, delivered before the College of Physicians in 1855, and from a review by Dr. Jenner, “On the Proximate Cause of Fever,” in *The British and Foreign Medico-Chirurgical Review* for 1856. Knowing how difficult it is to convey an orthodox account of the nature of fever; fully impressed with the great importance of the subject; and believing, as Dr. Jenner has expressed, “that so consistent a theory of the nature of fever, and one so largely supported by facts, has not been placed before the profession as that developed by Dr. Parkes, I only hope I may be able to do it justice in the



attempt to lay it before the student of medicine in the following form. In the eloquent language of Dr. Parkes, 'I shall have to allude to inexplicable phenomena, to vast spaces still unfilled by solid facts, to spots unknown to observation, and to regions lighted only by the dim and treacherous ray of speculation.'"

The practical object aimed at in the exposition about to be given, is to fix the scientific principles which ought to guide clinical investigation in determining the *Natural History* of fevers generally; and especially to define the differences which subsist among specific fevers; and so aid in determining the conditions under which fevers are generated or propagated—their development, course, or progress, and their defervescence.

"A hot skin, a quick pulse, intense thirst, scanty and high-colored urine," are phenomena common to many diseases; and when they are present it is said that the patient is *feverish*, or suffers from *fever* or *pyrexia*. There are some diseases in which such symptoms constitute the prominent, and almost the only appreciable phenomena, and which run a more or less definite course, without the necessary development of any constant local lesion. Such diseases have been emphatically termed "fevers," or sometimes *specific*, *primary*, or *idiopathic fevers*. When diseases marked by local lesions—such, for instance, as the local inflammations—are attended by the symptoms just stated, then the *pyrexia*, *fever*, or *feverish* symptoms which attend them, are said to be *secondary* or *symptomatic*; and the physician is accustomed, when he deals with such cases, to abstract the symptoms of fever from the other symptoms proper to the special affection. In other words, he prescribes for, and tries to cure the special affection, and not the *fever*, because he knows that when he has subdued the local disease the *fever* will subside. Not so, however, with the fever of a specific disease like *small-pox*, *typhus*, or *typhoid fever*: the physician cannot cure such a fever; but he may guide its course, by judicious management, as an experienced pilot may guide a ship and preserve it through a storm.

It is to the nature of *fever* considered in its abstract relations that the attention of the student is here directed, and not to any particular fever, such as *ague*, *typhus fever*, or the like. It is to *fever* in general that the following observations apply. It is to the *pyrexial symptoms* which are common to many diseases, such as to *small-pox*, *scarlatina*, *measles*, *typhus*, *ague*, *pneumonia*, *nephritis*, *meningitis*, and which, "like shadows to substance, are necessary to the very existence of such diseases, but yet are not, *per se*, any one of these diseases."

Galen defined fever as a preternatural heat—" *Calor præter naturam*." Subsequently many other additional clauses were added to this definition, such as "quick pulse," "turbid urine," and the like; but still the improved definition would not meet the requirements of every case; and now it is fully recognized that of all the clauses and phrases in the usual definitions of fever, "preternatural heat" is the only one whose accuracy is unimpeachable. In all cases, therefore, where fever is present, there are two points to be determined; namely,—(1.) *The amount of the preternatural heat by accurate*



measurement; (2.) *The amount of the tissue-change*, as represented by an estimation of the relative amount of all the excreta to the body weight.

It is the exact sequence of phenomena we desire to know in every case where pyrexia is present, as well as the meaning and co-relation of the phenomena: and usually symptoms sufficiently characteristic become developed and superadded to the febrile phenomena, by which the physician is able to define the specific nature of the disease or fever as a whole, and to say of this case or of that, "It is a *typhoid fever*," or "It is an *ague*," or "It is a *rheumatic fever*," or "a *pneumonia*," or "a *dysentery*," or any other form of illness where pyrexia is present, which we are able clinically to recognize. It is not very long since we were able to do this. Up till within a comparatively short time ago, the classification and diagnosis of "Fevers" was not such as to distinguish and separate their varied forms and varieties from each other. "Common continued fever," for example, was a comprehensive name which included many very different types of fever; and no means of observation have been of late so exactly discriminating, so as to distinguish one form of disease from another where fever coexists, as accurate observations on the temperature of the patient, determined by the thermometer. In acknowledging this great fact, it is important to observe that the absence of such exact observation, and the trusting to general signs alone, have hitherto led to great confusion—a confusion which has been most unjustifiably and unfortunately increased by a pernicious system—becoming too common—of naming "Fevers" from the place or locality where supposed varieties of fever have prevailed as epidemics; or by the use of local or provincial native names. For example, the Walcheren Fever, Levant Fever, Mediterranean Fever, Crimean Fever, Bulam Fever, African Fever, Fernando Po Fever, Lisbon Fever, Bengal Fever, Pucca Fever, Gall-sickness of the Netherlands, Hong Kong Fever, and other names not less barbarous, may be quoted. Except as matter of history, and as beacons to warn us from the danger to science, let these and such-like names be consigned to oblivion. With the exact means at the disposal of the physician as aids to diagnosis (and which are about to be described), every variety of illness where fever takes a part may be accurately distinguished, its type recognized, and its place fixed in nosology; or if it should be anomalous, its exact departure from the type may be not less accurately defined and described.

The phenomena which thus call for special investigation are those which are strictly related to the development and progress of the febrile state. They ought to be determined by clinical observation in all cases of disease where fever may be present. The facts to be ascertained are not less significant of the abatement, subsidence, or "*defervescence*" of the febrile state than of the advent of local lesions. The term "*defervescence*," in fever, is a comparatively new one in English pathology. It was first used by Professor Wunderlich, and subsequently adopted in this country by Dr. Parkes. It signifies the period during which the temperature of

the fevered body is declining to its normal amount from that intense degree of heat attained in the state of accession of the febrile phenomena. This "*defervescence*" may be sudden, when it is regarded as a "*crisis*;" or it may be gradual, and is then described as a "*lysis*"—the "*insensible resolution*" of the older authors; or it may be partly sudden and then slow, when it may be described as "*wave-like*," with gradual and sometimes regular alternations of high and low temperature, as Dr. Parkes was the first to point out (*The Composition of the Urine in Health and Disease*, p. 270).

**The Usefulness of the Thermometer at the Bedside in the Diagnosis of Pyrexia.**—One hundred and ten years ago (1754), Antonius de Haen, the first teacher of clinical medicine in the Hospital of Vienna, impressed his pupils with the necessity of attending to the temperature of the body in disease, *as measured by a thermometer*, instead of being judged of merely by the hand. He showed that even in the cold stage of ague, with the teeth chattering and the body shivering, the temperature of the blood is rapidly rising, although the pallid skin may really be colder than usual—its supply of blood being diminished by the contraction of the bloodvessels. He first demonstrated with *measured accuracy* how much the heat of the body is augmented under the influence of the febrile state; and when the crude appliances and the rough instruments of a hundred years ago are compared with the delicacy and refinements of "the instruments of precision" of the present day, it may be of interest now to observe how the progress of knowledge and the powers of modern research have not suffered the valuable pathological lessons to be lost sight of which are to be learned from the clinical use of the thermometer, as De Haen taught a hundred years ago. When the hand of the physician alone is used to judge of the temperature of a patient, or when the feelings of the patient are alone taken as a measure of his temperature, it can easily be understood how such kind of observation is extremely fallacious, doubtful, and unsatisfactory. The determination of the amount of heat in fever cases is stamped by a much more early appreciation of its importance and value than even since the time of De Haen; for, ever since the days of Hippocrates, the Physician and the Surgeon have been in the habit of applying the hand to the skin of the patient, to appreciate the presence of abnormal heat; but the practical application of the thermometer in place of the hand, while it is obviously a more accurate method, has never come into general use, mainly on account of the difficulty of getting instruments sufficiently sensitive and trustworthy—instruments, in fact, of sufficient precision. The time and trouble required to work with crude and inefficient instruments soon brought them into disuse and discredit; but now the instruments required may be obtained so delicate and accurate, and the time they take to apply them is so insignificant, that the student of medicine and the physician have no excuse for neglecting to use them. When it is remembered, also, that Galen's definition of fever is still the one whose accuracy remains not only unimpeachable, but fully demonstrated, and now recognized; that it describes fever to consist in "a preter-



often differing also very much from each other. If German thermometers are used, it is therefore necessary to compare the thermometer used with a standard one, and note the differences *between every degree*. A thermometer is bad, and all but useless, if the differences between various degrees are unequal, but is quite serviceable if the same sum is to be added or subtracted for *each* degree. The price of such an instrument is moderate; therefore it need not be difficult nor expensive for a student to acquire a competent practical knowledge of "the thermometry of disease."

As it is necessary to have a good thermometer, with a uniform and correct scale, having a range from  $88.2^{\circ}$  to  $110.7^{\circ}$  Fahr., exhibiting also 5ths Fahr. of degrees, I have arranged, in a convenient wooden case (with the aid of Mr. Casella, the accurate and careful instrument-maker at 23 Hatton Gardens, London), two thermometers graduated in Fahrenheit degrees, especially for the use of medical men. Each box contains—

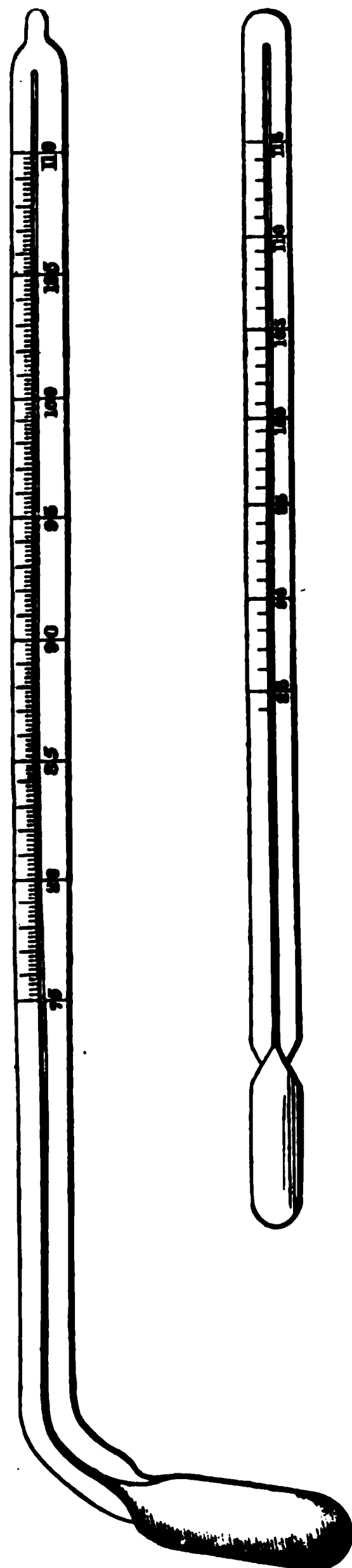
1. *An Ordinary but very Sensitive Thermometer*, Fig. A, made with a curve, in order that its bulb may be the more easily and perfectly fitted into the axilla, while the stem being carried upwards, renders the reading *in situ* more easy.

2. *A Straight Thermometer*, Fig. B, which, being a maximum self-registering one (known as "Phillip's maximum"), does not require to be read *in situ*, but may be removed from contact with the part, and read when convenient. Both thermometers are graduated up to at least  $112^{\circ}$  Fahr., and each degree is subdivided into *fifths*.

**Directions for Use.**—I. *The Curved Thermometer*, Fig. A. Its bulb must be well fitted into the arm-pit, being introduced below the fold of the skin covering the edge of the *pectoralis major* muscle, and so kept in close contact with the skin, completely covered and firmly surrounded by the soft parts. In very thin or very old persons this adjustment requires special care. The instrument must be retained *in situ* during a period of not less than *three minutes*; and the height of the mercury in the graduated stem must be read *while the thermometer is still*

FIG. A.

FIG. B.



One-half the real size.



In chronic cases, when febrile attacks and their concomitant dangers may be expected, as well as in acute cases, after return of the normal blood-heat *one* daily observation will be found sufficient. This single observation may be best made in the afternoon, or at that hour of the day in which generally some apparent change takes place.

It is advisable to induce nurses, friends, or other attendants on the sick (whenever they seem apt pupils), to make notes of any considerable excitement or restlessness, hot hands, increased heat of head, and to consult at once the thermometer. They may thus, perhaps, tranquillize the patient and his friends when the instrument does not indicate any material increase of heat; but the sudden appearance of any considerable increase of temperature would always be (as we have seen) a fact of vital importance.

It has been recommended by some to place the thermometer under the tongue, as the best place. On the contrary, the cavity of the mouth is the worst place in which the thermometer can be put, because the temperature there is continually varying according to the quantity and temperature of the air used in respiration; and if the atmosphere is cold, and deep inspirations are made, large differences may be observed, compared with the temperature in the axilla. Therefore it is necessary not to trust to observations made with the thermometer in the mouth.

In cases which do not require the most rigorous and extreme accuracy (as cases observed for the sake of scientific information require), *three to five* minutes is found quite sufficient in private practice for the application of the thermometer. The simplest and most convenient way is to heat the instrument before inserting it into the patient's axilla, just as the surgeon heats the catheter before he introduces it into the urethra. This may be done by holding the thermometer in the warm hand, or into water heated to a temperature of nearly  $30^{\circ}$  R. =  $99.5^{\circ}$  Fahr.: and, after the instrument is properly placed, be satisfied if *two observations at intervals of one to two minutes give exactly the same result*.

The rapidity with which the mercurial column rises depends on the degree of temperature present. The rapidity of the rise of temperature ought to be noted, as well as the maximum height. If the temperature be above the normal standard, a sensitive thermometer will indicate that fact within the first minute; and as the quickness of the rising depends upon the existing temperature, the physician is able, after some experience in the use of a particular instrument, to form an approximate judgment of the amount of rising of temperature to be expected in any particular case from the slowness or rapidity of the rise of the mercury after half a minute.

II. *Fluctuations of Temperature within the Limits of HEALTH; and the Co-relation of the Animal Heat with the Pulse and the Respiration.*

Several observers in Germany, France, England, and the Tropics, have now determined these fluctuations with great accuracy, so that ample and sufficient data are on record to furnish a standard for comparison in cases of disease.

The temperature of the body is the result of the opposing action

of two factors: 1st, Of development of heat from the chemical changes of the food, and by the conversion of mechanical force into heat, or by direct absorption from without; and 2d, and opposed to this, Of evaporation from the surface of the body, which regulates internal heat (Parkes's *Hygiène*, p. 432).

With reference to the normal range of temperature, our most trustworthy information is mainly due to Valentin and Traube, in Germany; to Edwards, Becquerel, Breschet, and Bernard, in France; and to Dr. John Davy, in England and the Tropics. All agree in stating that the ranges of temperature vary in different parts of the human body; but, as a general practical result, it is equally agreed that in temperate regions the normal temperature *at completely sheltered parts of the surface* of the human body amounts to 98.4° Fahr., or a few tenths more or less; and a rising above 99.5°, or a depression below 97.3° Fahr., are sure signs of *some kind* of disease, *if the increase or depression is persistent*.

Valentin proved by many experiments that all warm-blooded animals surrounded by an atmosphere of 50° Fahr. to 68° Fahr. have a temperature of about 99.5° in the back of the mouth, the rectum, or other accessible *internal parts*; and at *completely sheltered parts of the surface* it is a degree lower—namely, 98.4° or .5°. But the most common temperature is about 97.5°, according to Dr. Compton's more recent observations. Dogs have a temperature similar to that of men; so that a knowledge of thermometry in the diseases of animals will prove not less valuable in veterinary pathology than in human, and perhaps more so, inasmuch as such animals are deprived of speech to express their feeling.

The observations of Dr. John Davy, originally communicated to the Royal Society, are the most numerous and extensive in detail on record, which indicate the fluctuations of temperature within the limits of health. An abstract is given in the following table:

FLUCTUATIONS OF TEMPERATURE WITHIN THE LIMITS OF HEALTH (DAVY).

I.—IN TEMPERATE REGIONS (ENGLAND).

Period of Day.	Temperature of Body.	Pulse per Minute.	Respiration per Minute.	Temperature of Room.
Morning, 7-8,	98.74° Fahr.	58	16	50.9° Fahr.
Afternoon, 3-4,	98.52° "	55	15	54.7° "
Midnight, 12,	97.92° "	55	15	62° "

II.—IN TROPICAL REGIONS (BARBADORS).

Morning, . . 6	98.07° Fahr.	54	14	76.7° Fahr.
Afternoon, . . 2	98.9° "	56	15	83.6° "
Night, . . 10-11	99.0° "	60	15	79.8° "

The general result may be stated as follows:

1. IN TEMPERATE CLIMATES the *Maximum temperature* is in the



early morning after waking: it fluctuates till nightfall, and is lowest about midnight; average difference,  $0.82^{\circ}$ .

2. IN TROPICAL REGIONS the *Minimum temperature* is in the early morning after waking: it fluctuates, and is highest during the day.

3. *Average temperature* throughout the year,  $98.4^{\circ}$  Fahr.,—the temperature of the air averaging  $55.5^{\circ}$  Fahr.

The observations of Dr. Davy and of Edwards have shown that the amount of animal heat may be considerably altered by a number of collateral circumstances. But the great distinction between these alterations of temperature in health, and those which are the result of disease, is, that these variations are generally temporary, and within narrow limits—amounting to mere fractions of a degree—rarely more than from  $1.8^{\circ}$  Fahr. to  $3.6^{\circ}$  Fahr. (VALENTIN and DAVY), whereas those which are due to disease are persistent so long as the disease exists.

The following are the collateral circumstances which mainly influence animal heat in our daily life, and which require to be remembered in order that erroneous conclusions may not be drawn: (1.) *Active exercise* (not carried to the extent of exhausting fatigue) raises the temperature proportionally to the degree of muscular exertion made. (2.) *Exposure to cold* without exercise lowers the temperature. (3.) *Sustained mental exertion* reduces temperature about half a degree. (4.) The amount of heat is also at first reduced after a *full meal* and the use of alcohol; but it rises again as digestion advances. (5.) There are diurnal fluctuations capable of being thus determined. (6.) The temperature of the body rises with the temperature of the air; and sudden transitions from a cold to a hot climate induce a feverish state marked by increase of temperature, accelerated action of the heart, and quickened respiration, especially on bodily exertion. (7.) The average temperature within the *tropics* is nearly  $1^{\circ}$  Fahr. higher than in temperate regions. (8.) The temperature is more readily and rapidly affected—more sensitive, so to speak—than either the pulse or the respiration; and this is especially the case in disease.

The amount of abnormal increase of temperature is usually proportionate to the degree of frequency of the pulse, and to the other signs of general disease. Yet such congruity of phenomena is sometimes in part or wholly absent or incomplete; and in the cases in which a disproportion or incongruity exists between the increase of temperature and the pulse or other febrile phenomena, it is the accurate measurement of the temperature which is most of all to be relied upon. As a general rule the co-relation of pulse and temperature may be stated as follows, namely: “An increase of temperature of ONE DEGREE above  $98^{\circ}$  Fahr. corresponds with an increase of TEN beats of the pulse per minute, as in the following table:

Temperature of	$98^{\circ}$	.	.	.	Corresponds with a pulse of	60
“	$99^{\circ}$	.	.	.	“	70
“	$100^{\circ}$	.	.	.	“	80
“	$101^{\circ}$	.	.	.	“	90
“	$102^{\circ}$	.	.	.	“	100
“	$103^{\circ}$	.	.	.	“	110
“	$104^{\circ}$	.	.	.	“	120
“	$105^{\circ}$	.	.	.	“	130
“	$106^{\circ}$	.	.	.	“	140 ”





course, terminating in death, as certainly and as rapidly as if indicated by the ordinary train of symptoms. Discovering then, as it were by accident, that the temperature in this lunatic was as high as  $104.5^{\circ}$  Fahr., and that his pulse was rapid, Dr. Davy's attention was more particularly aroused; and although the man made no complaint, but had a good appetite, with his digestive functions, so far as was known, acting well, yet disease of the lungs was thus discovered. The lunatic died in a month, of acute tuberculosis, not otherwise expressed by symptoms beyond the great, persistent, and continuous elevation of temperature thus incidentally noticed. There were ulcers of the *larynx* found after death, but there had been no affection of the voice; there were *vomicæ* and *tubercles* in the *lungs*, but there had been no cough; there were *ulcerations* of the *intestines*, but there had been no diarrhœa; there was disease of the *testes*, *vesiculæ seminales*, and *prostate*, of a severe kind, but these lesions had been equally latent during life, except hardening and enlargement of the testicle without pain,—all which conditions were only casually observed.

In this very instructive case, a temperature of *six degrees* Fahr. above the normal standard was the earliest indication of disease (*Researches, Physiological and Anatomical*, vol. i, p. 206).

But it is mainly to Wunderlich, the Professor of Medicine in Leipsic, that we are indebted for an elaborate exposition and persevering advocacy of the usefulness of daily records of the temperature of fever patients, and the constant employment of the thermometer as a means of diagnosis at the bedside. On this subject he has written much, from an extensive experience, embracing at least half a million exact thermometric observations, following the continuous progress of individual diseases, the results of which he has compared in more than 5000 patients. He constantly employs the thermometer in his private practice, and bears unqualified testimony to its sterling value in the early detection of disease, and as often furnishing an important guide to treatment. When the physician once becomes accustomed to the investigation of disease by the thermometer, he regards its daily employment as indispensable, for it imparts a certainty to his observations, attainable by no natural penetration, and which no other method of investigation can convey (*Medical Times and Gazette*, June 19, 1858, and September 28, 1861).

More detailed results are published by the assistants or pupils of Wunderlich, in the *Archives für Physiologische Heilkunde*, 1860, p. 385, and 1861, p. 433; and the principal conclusions have been summed up by Wunderlich himself, in his *Handbuch der Pathologie*. From these sources the information given in this and former editions of this text-book was originally compiled.

Wunderlich gives some striking instances of disease being indicated by thermometric observation before it could be detected by any other means:

*In ague*, several hours previous to the paroxysm, the temperature of the trunk of the patient's body begins to rise; and when the disease seems to have disappeared, an increase of temperature may be

detected periodically, unaccompanied by any other symptom. So long as this periodic rise of temperature continues, the patient is only apparently, but not really cured.

*In typhoid fever*, during the exacerbations especially, the rise of temperature or its abnormal fall will indicate what is about to happen three or even four days before any change in the pulse, or other sign of mischief, has been observed. A sudden and marked reduction of temperature has thus denoted hemorrhage from the sloughs of Peyer's patches in typhoid fever, several days before it appeared in the stools. A case of this kind is recorded by Dr. Parkes. It occurred in a female twenty-five years of age. Diarrhœa was considerable, and blood was largely passed in fluid stools the night before the 17th day of the fever. On the morning of that day the temperature was as low as 93° Fahr., rising in the evening to 101° Fahr.

It is rare, however, that a definite diagnosis or prognosis can be based on a single observation; but sometimes it may be sufficient, as in the following instances:

When the temperature is increased beyond 95.5° it merely shows that the individual is ill, and suffering from some disease, and that when considerably raised, as with a temperature of 101° to 105°, the febrile phenomena are severe; that when a great height is reached, as at temperatures above 105° Fahr., the patient is in imminent danger; and that with a rising temperature above 106° Fahr., to 108° or 109° Fahr., a fatal issue may almost without doubt be expected in a comparatively short time. The highest temperatures before death have been observed in cases of scarlet fever and of tetanus.

A definitive diagnosis may also be based on a single observation, under the following circumstances:

A person who yesterday was healthy, but exhibits this morning a temperature above 104° Fahr., is almost certainly the subject of an attack of ephemeral fever or of ague; and should the temperature rise up to or beyond 106.3° Fahr., the case will certainly turn out one of ague, or some other form of malarious fever.

A girl eighteen years of age, supposed to be suffering from hysteria, but simulating a case of cerebro-spinal meningitis. A temperature of 103.5° confirmed the diagnosis of meningitis and negatived that of hysteria. The case terminated fatally (COMPTON). Again, in a patient whose temperature rises during the first day of illness up to 106° Fahr., it is certain he does not suffer from typhus nor typhoid; and of a patient who exhibits the general typical signs of pneumonia, but whose temperature never reaches 101.7° Fahr., it may be safely concluded that no soft infiltrating exudation is present in the lung.

Again, if a patient suffer from measles, and retains a high temperature after the eruption has faded, it may be concluded that some complicating disturbance is present.

Single observations with other means of diagnosis will often determine whether the disease is one of danger or not.

In typhoid fever a temperature which does not exceed on any

evening  $103.5^{\circ}$  Fahr. indicates a probably mild course of the fever—and especially if the increase of temperature takes place moderately, towards the beginning of the second week. A temperature of  $105^{\circ}$  Fahr. in the evening, or of  $104^{\circ}$  Fahr. in the morning, shows that the attack is a severe one, and forebodes danger during the third week; on the other hand, a temperature of  $101.7^{\circ}$  Fahr. and below, in the morning, indicates a very mild attack, or the commencement of convalescence. In pneumonia a temperature of  $104^{\circ}$  and upwards indicates a severe attack. In acute rheumatism a temperature of  $104^{\circ}$  is always an alarming symptom, foreboding danger, or some complication such as pericardial inflammation. In a case of jaundice otherwise mild, an increase of temperature indicates a pernicious turn. In a puerperal female an increase of temperature indicates approaching pelvic inflammation. In tuberculosis an increase of temperature shows that the disease is advancing, or that untoward complications are setting in.

In short, a fever temperature of  $104^{\circ}$  to  $105^{\circ}$  Fahr. in any disease indicates that its progress is not checked, and that complications may still occur.

But it is by continuous daily observations that the most important results have been arrived at, especially in the hands of Wunderlich, Greisinger, Traube, Billroth, Parkes, Jones of Augusta, Ringer, and others who are now working most actively in this field of labor.

Certain febrile diseases have been found to have typical ranges or daily fluctuations of temperature throughout their course. In pure unmixed and uncomplicated cases this is found to be so constant that the differential diagnosis may be established by accurate observation of the temperature continuously from day to day. This has now been determined, especially in cases of malarious fever, typhus, typhoid fever, small-pox, scarlatina, measles, rheumatism, pyæmia, pneumonia, acute tuberculosis. In each of these diseases the temperature is one of the most certain (although not the only) means for determining the real state of the patient as regards morbid disturbances or complications, and a careful observation of temperature from day to day is indispensable for judging as to the prognosis. Frequently it affords the best and ultimate means of deciding in doubtful cases, and often it is the best corrective of a too hasty conclusion: for example, the characteristic variations of the temperature in a typical case of enteric, intestinal, or typhoid fever, are of such a kind that they are not found in any other disease. Intestinal catarrh, severe forms of pneumonia, malarious fever, acute tuberculosis, meningitis, some stages of Bright's disease, may each simulate typhoid fever, and may exhibit some of its most characteristic symptoms; but observation with the thermometer as to the patient's temperature from day to day, will at once, or after a very few days, establish the distinction with certainty.

In the course of many diseases, whose diagnosis has been accurately determined, if the temperature departs from its normal or typical range, the thermometer will furnish the best and the earliest

indication of any untoward event, such as the additional development of disease, or other visceral complications in its course.

When once the typical range of temperature (*normal*, as it were, of the particular disease) is determined, a basis is laid for appreciating irregularities or complications in its course in particular cases. For example, a patient exhibits symptoms of fever of the typhoid type, but during the progress of the first week his temperature becomes normal, for however short a space of time;—the occurrence of this event proves that the fever is not what it was supposed to be. Again, a patient may suffer from all the general symptoms of incipient pneumonia; but there still is a doubt as to whether infarction of the lung has taken place. The sputa being suppressed, or not procurable, does not assist the diagnosis. If, however, the temperature is found to be normal, it is certain that no croupous exudation has taken place in the lung, and that there is no pneumonia. Again, if a tuberculous patient has a sudden attack of hæmoptysis, and if the temperature of his body is normal during and subsequent to the attack, no reactive pneumonia, nor any exacerbation of the tuberculous exudation need be expected. This is a new field open for investigation in cases of phthisis.

Again: In all cases of convalescence, so long as the defervescence proceeds regularly as measured by the temperature, no relapses need be feared: on the other hand, delayed defervescence in pneumonia, the persistence of a high evening temperature in typhus or typhoid fever, or the exanthemata, and the incomplete attainment of normal temperature in convalescence, are signs of great significance. They indicate incomplete recovery, supervention of other diseases, unfavorable changes in the products of disease, or the continuance of other sources of disturbance requiring to be carefully examined into. The onset of even a slight elevation of temperature during convalescence is a warning to exercise careful watching over the patient, and especially for the maintenance of a due control over his diet and actions.

Continuous daily observations by the thermometer thus teach the typical laws of particular forms of fever, and supply the grounds or basis by which it is determined whether any individual case is progressing as it ought to do. Such knowledge can only be acquired by repeated observation of numerous cases; and deviations from the normal temperature in certain diseases are stable in proportion to the typical character and full development of the particular disease. But even in such diseases we may have an increase or decrease of temperature proper to the disease brought about by *accidental* influences. Such instability, however, is only temporary, and of short duration, when the *accidental* influences act but *transitorily*. For example, the temperature proper to the disease may be lowered under the influence of a profound sleep, bleeding, epistaxis, the relief of constipation or of the retention of urine, and the like; or it may be raised after excitement of a mental kind. But any such alterations, unless they are dependent upon a change in the disease-process itself, will become effaced after twelve or twenty-four hours at the most, when the temperature again resumes the typical

character diagnostic of the particular disease. In continued fevers the temperature is generally less high in the morning than in the evening.

Stability of temperature from morning to evening is a good sign; on the other hand, if the temperature remains stable from evening till the morning, it is a sign that the patient is getting or will get worse.

When the temperature begins to fall from the evening to the morning, it is a sure sign of improvement; on the other hand, a rise of temperature from the evening till the morning is a sign of his getting worse.

When it is found, in a bad case of typhoid fever, that some morning about the third week the temperature has fallen to  $99.5^{\circ}$ , the reparative stage has begun—the healing of Peyer's patches; and when a similar fall of temperature is observed in the evening, convalescence has commenced.

In pneumonia, when a marked fall of temperature occurs in the evening, the period of crisis has arrived.

In measles, when the maximum severity of the eruptive stage has been reached, the temperature falls.

A sinking from a considerable height down to a normal temperature suddenly (within twenty-four hours), occurs in a few exanthemata,—measles, variola, rarely in pneumonia, typhus, and pyæmia.

In tuberculosis, especially in its acute form, the persistent maintenance of a uniformly high temperature will alone show that no arrest in the progress of the disease has occurred.

The correlation of pulse, respiration, and temperature is of great importance to be determined in many acute diseases; and especially in pneumonia, if the mean of the temperature is not above  $104^{\circ}$  Fahr., and that of the pulse is not above 120 in a minute, and the mean of the respirations not over 40 in the same time, the case must be considered a slight one; and if the patient is otherwise healthy, he will surely begin to get well in from eight to twelve days, without any medical treatment beyond attention to antiphlogistic regimen.

Convalescence is known to commence when the disease-process ends; and this precise point can only be fixed by continuous thermometric observation. The morbid process does not end till the normal temperature of the body returns, and maintains itself unchanged through all periods of the day and night.

Regularly continuous observations of the temperature exhibit the precise point at which the disease-process terminates, and the degree of its complete development. When this point has been determined on, a retrospective view may be taken of the character of the disease, as to the purity of its typical form or its complexity, and a prognosis may be hazarded as to the probability or doubtfulness of recovery. The morbid process has not terminated till the normal temperature of the body returns, and remains unchanged in the evenings and throughout all periods of the day. The transition from the febrile state into defervescence is either slow (lysis) or rapid (crisis). A regularly continuous defervescence is always a sure sign of convalescence. Its occurrence will save other investi-





(2.) The temperature may become more or less moderated, while the pulse is increased in frequency, and the other symptoms become more and more threatening. Such diminution of temperature, amidst conditions which do not harmonize with it, must be regarded as a pretty certain sign of approaching dissolution.

But, on the other hand, there are cases in which the observation of the temperature yields the most favorable signs for prognosis. For example, when it is found, in a bad case of *typhoid fever*, that the temperature has fallen some morning to  $30^{\circ} \text{R.} = 99.5^{\circ} \text{Fahr.}$ , we know that the reparative stage is entered upon; and when a similar fall of temperature is observed in the evening, convalescence has commenced. In *pneumonia*, when a marked fall of temperature occurs in the evening, it shows that the period of crisis has arrived. When the temperature falls in *measles*, the maximum severity of the eruptive stage has been reached; and when, in the first stage of *variola*, we observe a quick return to the normal temperature, we may feel certain that a slight form of the disease, free from danger, is likely to ensue.

A decrease of temperature below the normal heat is rare. It happens sometimes transitorily, announcing thereby a favorable crisis, by preceding the return to a normal temperature. It is also met with sometimes during the morning remission of *remittent fever*; also during the apyrexia of *intermittents*; in acute collapse, preceded or not by fever; in chronic wasting diseases; and sometimes, also, on the approach of death, especially in typhus fever.

A remarkable inequality in the distribution of the temperature over different parts of the body (face, hands, feet, etc.) may occur during the shivering preceding fever, in collapse, and in the agony of approaching dissolution. Sometimes, also, such unequal distribution may occur in disorders of the chest and abdomen, in some local skin diseases, and in partial paralysis. This fact is not of importance or utility, for diagnosis or prognosis; but it requires to be known, in order that erroneous conclusions may not be drawn.

IV. *Of the Ranges of Temperature in Diseases where Fever is present, as related to the Amount of the Excreta.*

The particular degree of heat and the waste in every febrile disease are represented by—*something*. The physician sees the fevered patient wasting before his eyes. Every tissue is wasting, and, in correlation with the excessive generation of heat, how is this waste expressed? As a rule, it is expressed by the amount of *excreta*.

To Dr. Parkes in this country, to Dr. Jones of Augusta (in cases of malarious fever), to Virchow and Wunderlich in Germany, is the merit mainly due of having demonstrated, by clinical and experimental observation, that “the morbid development of heat, as measured by a thermometer, is associated in some cases with MORE abundant, in other instances with LESS abundant *excreta* from the body than in health; that the temperature and the amount of the excretions bear some undetermined relation to each other; and that the loss of weight of the patient is due to increased and rapid elimination of material with increased tissue-change, associated with the increase of temperature.”



So far as physiological facts have elucidated the normal generation of heat in the healthy body, so far has the abnormal generation of heat essential to the *febrile* state been clearly made out. In health the normal temperature produced by chemical change in the body, is represented in the excretions by so much *urea*, *sulphuric acid*, *carbonic acid*, *excretive volatile acids* of the skin, and the like; but in the febrile body a higher temperature is represented in the excretions, in some cases by a larger, and in others by a smaller quantity of *urea*, *sulphuric acid*, and *probably carbonic acid*\* (PARKES).

The most opposite statements have thus been made regarding the amounts of the excretions in fever, compared with the quantity excreted in health; and at present many excellent observers hold that these excretions are always, and of necessity, increased; others, no less exact, affirm that they are invariably, or almost always, diminished. Such discrepancy of statement is due, in the first instance, to the difficulty of collecting and measuring exactly the amount of all the excretions. "Two of the excretions, the cutaneous and the pulmonary, cannot be collected and measured with anything like the accuracy necessary in such an inquiry: even in health, such an inquiry is difficult, and in fever it is almost impossible." By careful and accurate observation at the bedside, however, Dr. Parkes has been able to obtain very close approximative data to found his conclusions upon, relative to the increase or diminution of the excretions. He assumes that when the respirations are not quickened (*i. e.*, about eighteen times in a minute, or about one act of respiration for every four beats of the pulse), and when the skin is not evidently sweating, the excretions by these two organs are not increased; and, on the other hand, an increased excretion by these organs may reasonably be inferred, if the exercise of their function is unusually active, and if there are tolerably copious perspirations. The other two excretions, namely, the urine and intestinal discharges, can be measured with accuracy, and the urine in particular is a valuable index of the metamorphoses of tissue. The *urea* alone represents two-thirds of the whole quantity of *nitrogen* which passes off; the *sulphuric acid* (the sulphates of the food being

---

\* In the study of special diseases, the student ought frequently to estimate the quantity of *excreta* by the urine, as one of the best methods for enabling him to appreciate the changes which go on in the body during disease. To aid him in prosecuting such researches, he is recommended to consult the work of Dr. Parkes, *On the Composition of the Urine*, and to follow the directions given on the *Examination of the Urine*, towards the end of the second volume of this text-book, for obtaining quantitative results by the volumetric method.

Average quantity of urine passed in twenty-four hours, .				52½ to 56 ounces.
Average amounts of solids				945 grains.
"	"	<i>Urea</i>	"	512 "
"	"	<i>Chlorine</i>	"	126.76 "
"	"	<i>Free Acid</i>	"	33 "
"	"	<i>Phosphoric Acid</i>	"	48.80 "
"	"	<i>Sulphuric Acid</i>	"	31.11 "
"	"	<i>Uric Acid</i>	"	8.5 "
Specific gravity. . . . .				10.20.

accounted for) represents almost entirely the oxidation of sulphur; and the oxidized phosphorus of the body passes out in great measure, though not altogether, as urinary phosphoric acid. Therefore a careful examination of the urine, and of the intestinal discharges, with an approximative estimate of the pulmonary and cutaneous excretions, give sufficiently extensive and accurate materials for the question at issue.

The products excreted are thus of such a kind as to be eliminated, some by *the lungs*, some by *the skin*, some by *the bowels*, and some by *the kidneys*, and rarely by two or more modes of excretion—for when the discharges from the *skin* or *bowels* are profuse, those by the *kidneys* are deficient, as in the last two cases recorded in the following Table I, in which the augmented excretions are printed in italics. The facts thus so carefully observed by Dr. Parkes, confirmed by Alfred Vogel, Heller, and others (but chiefly in regard to the excretion of urea only), justify the conclusion—“*That increase of temperature may be attended with increased elimination; and therefore presumably with increased tissue-change.*”

TABLE I.—ABSTRACT OF CASES OBSERVED BY DR. PARKES IN WHICH SOME OF THE EXCRETIONS ARE INCREASED IN CONSEQUENCE OF THE FEBRILE STATE.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Rheumatism.	Fahr. 8°.	Not noted.	<i>Sweating profusely.</i>	Discharge as usual.	Solid matter excreted <i>greater than in health by 100 grs., and due to urea and sulphuric acid.</i>
Rheumatism.	Fahr. 8°.	Not noted.	<i>Sweating profusely.</i>	Discharge not diminished.	<i>Solid matters excreted greater than in health by 200 grs., and due to urea and sulphuric acid.</i>
Typhoid Fever.	Several degrees.	<i>Rapid.</i>	<i>Moist.</i>	Not increased.	<i>Increased by 60 grains.</i>
Erysipelas of Head and Face.		<i>Quick.</i>	<i>Moist.</i>	Unaltered.	<i>Considerably augmented.</i>
Febricula.	Fahr. 8°.	Normal.	<i>Enormously augmented.</i>	Confined.	Solids less than normal by 91 grains.
Typhoid.	Fahr. 8°.	Not noted	<i>Sweating and Sudamina.</i>	<i>Diarrhœa profuse.</i>	Solids less by 78 grains.

TABLE II.—CASES OBSERVED BY DR. PARKES IN WHICH THERE WAS DIMINUTION OF THE EXCRETIONS.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Bronchitis of both lungs.	Fahr. 2.6°.	20 Respirations per minute.	Not increased.	Not increased.	Less by 112 grains.
Pneumonia acute sthenic.	Fahr. 5°.	80 per minute; expectoration scanty.	Slightly moist.	Confined.	Less by 220 grains.
Typhoid Fever.	Considerable.		No sweating.	No diarrhoea.	Below normal amount.
Acute Rheumatism.	Fahr. 4°.	Tranquil breathing.	Inconsiderable.	Bowels quiet.	Very small amount of urinary solids.

The abstract given in Table II shows that another conclusion is equally legitimate, namely—“*That the products of metamorphoses, as judged of by the excreta, may be diminished in febrile cases; and these apparently discordant statements are capable of being explained in various ways. In the first place, it is evident that more chemical change may go in the body than is represented by the excreta. The metamorphosis of blood or of tissues may not be carried to the point of forming those principles which can alone pass through the eliminating organs. A vast amount of imperfectly organized compounds may be formed and retained in the system, circulating with the blood or thrown upon certain organs.*” *Thus there may be increased metamorphosis with lessened elimination.* Several pathological facts point to such a conclusion.

1. It is in such febrile cases, with diminished excreta, that, at a later period of the disease, copious discharges from one or other of the eliminating organs occur. Thus, in the case of pneumonia referred to in Table II, severe spontaneous diarrhoea came on; and many other cases are quoted, with similar diminution of the excretions at the period of increased febrile heat, in which violent purging, sweating, or diuresis, with increase of urea and of sulphuric acid, subsequently occurred. Such discharges occurring during the progress and towards the termination of a febrile disease have been termed *critical*, the occurrence being called a *crisis*; and the particular day on which it happens, counting from the day of seizure, has been called a *critical day*. The term *crisis* or *critical* is applied because the occurrence of such discharges is usually coincident with more or less sudden fall of temperature, and general improvement in the condition of the patient, whose convalescence dates from the

critical day ; when, in common language, his disease is said to have "*got the turn.*" In such cases, therefore, a large amount of partially metamorphosed substances are retained until they are suddenly discharged, and the system freed from the noxious compounds. Coincident with the critical discharge, the temperature is found to fall.

2. But in another class of febrile cases retention of the products of metamorphosis is not followed by such a fortunate *critical* issue. At a later period in the history of some febrile cases, with diminished excreta, it is not uncommon for *secondary inflammatory* affections to occur, as if the blood were more contaminated; and it is sometimes observed that in a patient whose excreting organs are acting copiously, there occurs a diminution of excretion when a simultaneous or subsequent development of local disease becomes manifest.

The following Table of Cases recorded by Dr. Parkes is interesting from the exactness of the observations and the coincidence of the local lesions with suppression and retention of excreta during febrile states:

TABLE III.—TABLE OF CASES OBSERVED BY DR. PARKES TO SHOW LOCAL LESIONS COINCIDENT WITH SUDDEN RETENTION OF THE EXCRETIONS IN FEVER.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Rheumatic Fever.  Observed on the fifth, sixth, and seventh day of the disease.	Fahr. 2°.  Fahr. 2°.	No record.	Sweating profuse.  Lessened on the eighth day.	No intestinal discharge.	While 400 grains more than in health were being daily excreted, <i>suddenly</i> on the eighth day a diminution of the solids took place by 602 grains; and coincident with this diminution a local lesion became developed ( <i>angina faucium</i> ). Next day the excretion augmented, and the local affection subsided.
Typhoid Fever.	Fahr. 5°.  Fahr. 5°.	No record.	Great sweating.  Lessened much.	Diarrhœa profuse.  Diarrhœa ceased.	While the average daily excretion for eight days was 422.848 grains, a gradual diminution continued for three days, to the daily extent of 78 grains, when pleurisy came on.



The largest amount of *sulphuric acid* recorded by the same observer, when no medicine was taken, was in a case of rheumatic fever. It amounted to 52.668 grains; and under the influence of liquor potassæ in the same disease, he has known this excretion rise to 70 grains—more than twice as much as in health. The largest amount of *uric acid* excreted during a febrile disease in twenty-four hours, as recorded by Drs. Parkes and Garrod, has been 17.28 grains.

“The amount of tissue destroyed in order to furnish such quantities of excreta must be enormous; and if it is recollected that little or no food is taken by the *feverish* patient, and, therefore, that no materials are supplied for the reconstruction of the textures thus melting away three times more quickly than in health, the rapid loss in weight in fever, and the impaired nutritive condition of every organ at its close, will be at once evident.”

It is not yet determined where the increased destruction of the albuminous textures takes place; that is, whether it occurs in the blood or in the organs themselves. It is only known that both the albumen and the red corpuscles of the blood are lessened in amount at the end of a febrile disease; and of the various tissues none appear to waste so fast as the muscles, and especially the involuntary ones (*e. g.*, the heart in typhus fever). The fat of the body is rapidly absorbed in fevers; and Virchow asserts that the bones also become lighter. While it is known that much of the metamorphosis of these tissues takes place in the normal way, it is also probable that there is an unhealthy or perverted metamorphosis which leads to the appearance of compounds in the excretions, either altogether foreign to the body or foreign in respect of place and time. There is evidence of this in the peculiar smell of the perspiration, in the peculiar coloring matter of the urine, as well as in the occasional excretion by it of hippuric as well as of lactic, valerianic, and other organic acids.

Next to the occurrence of preternatural heat in fever, the *excessive retention of water in the febrile system* is perhaps the most remarkable and constant phenomenon. Notwithstanding the large amount of water frequently taken to quench the extreme thirst, the quantity of the urine is lessened, and is even scantiest when the skin is driest; and the “concentration of the urine appears to be almost as good an index of the amount of fever as the temperature itself.” The excretion of water by the skin is, as a rule, diminished; and it is a well-known clinical fact that the skin is drier than usual in febrile affections. Very early in the febrile state the buccal mucous membrane also becomes sticky, and the amount of saliva diminishes; and the decrease in the quantity of the gastric fluid during fever has been proved by the well-known experiments of Beaumont on Alexis St. Martin. The intestinal juices, like the gastric, are also probably diminished, for the stimulus of food is taken away, constipation prevails, and the fæces are dry (PARKES).

This retention of water in the system cannot at present be explained; but Dr. Parkes has suggested that it may possibly be due to the presence in the blood (or tissues generally) of some interme-



Virchow, in his definition, states that the essential phenomena *must* have their immediate cause in changes of the nervous system.

It is very difficult to substantiate this position, but the following general results prove the great influence of the nerves in febrile affections. Taken individually, they, no doubt, will impress different minds with different degrees of force, while collectively they cannot fail to furnish an argument in favor of the essential participation of the nervous system in fever:

1. There is the generally received physiological law, that nerves regulate the metamorphosis of tissue and the production of heat, both of which are altered in fevers (HELMHOLTZ, LUDWIG, BERNARD).

2. There are those experiments on the vagus nerve which bring about febrile phenomena, such as increased cardiac action, pulmonary congestion, anorexia, and nausea (BERNARD, PAVY).

3. There are those arguments derived from the various symptoms which announce, accompany, or terminate fever. (a.) The remarkable depression, apathy, sense of exhaustion and debility which usher in the febrile state. (b.) The shiverings, the contraction of the superficial vessels and of the skin. (c.) The increased rapidity of the heart's action, and the relaxation of the vessels, which soon follows the stage of contraction just noticed, or occurs without it. (d.) The congestion of the lungs. (e.) The periodicity of some of the phenomena of fever, and the occurrence of death or recovery on so-called critical days. (f.) The abnormal state of the secretions.

4. The fearfully rapid death which sometimes ensues in the early stage, from some unknown cause, may with justice be referred to profound nervous lesions; for there is great prostration, a galloping and early-failing pulse, and an excessively rapid respiration.

5. The effect of certain remedies, such as quinine, upon periodical febrile phenomena.

**Conditions which combine to produce the Complex Phenomena of Fever.**—1. The entrance into the blood of a morbid agent, and the alteration of the blood to a certain extent under its influence, come first in the order of events. Perhaps this occurs under the incubative period, when often there is no rise of temperature, no fever; that is, when no appreciable alteration of the general health can be discovered. The nature of the change in the blood is unknown.

2. When the change in the blood has reached a certain point, the nervous system, or rather that part especially connected with nutrition and organic contractility, begins to suffer changes in composition, which probably paralyze, impede, or destroy the normal molecular currents. When this occurs, the nervous symptoms of weakness, depression, rigors, contraction of some parts and vessels speedily followed by relaxation, mark the stage of invasion.

3. Various parts simultaneously, especially the muscles, and probably some of the organs, deprived in greater or less degree of nervous influence, begin rapidly to disintegrate, and by their disintegration preternatural heat is produced. Little or no fresh material is assimilated to compensate for the loss; great muscular prostration ensues; and destruction of tissue is increased by the accelerated action of the heart.





certainty better than the advances made in our knowledge of the "thermometry of disease," and the correlation of temperature with other morbid phenomena. By numerous careful observations it is now clearly established that the determination of the correlation of the pulse, the respiration, and the temperature of the body in disease is of the greatest practical importance, and especially when regarded in relation to the *excreta*. By such observation the natural course and termination of many diseases can be predicated with great certainty; and so our knowledge becomes more exact as to the nature and treatment of diseases. The pulse, the respirations, and the temperature, all and each of them, represent forces at work in the living body, all of which are capable of being measured with great exactness; and such measurements show how closely such expenditure of forces is related to the *excreta*, which represent the waste of our tissues in health and disease. The student or physician who continues to disregard the aid of thermometry in the diagnosis of febrile disease, or the military medical officer who ignores its value in the appreciation of feigned diseases, such as rheumatism, may be compared to the blind man guiding himself. By means of great practice and intelligence, the blind man will often proceed rightly; but the advantages of being able to see clearly are proverbially above all price. The necessity of using the thermometer will also soon become known to the general public, and patients will become dissatisfied if all known means of investigation are not employed in appreciating the nature of their malady. For many years the German student and physician has been familiar with its use; but, with the exception of Dr. Parkes, and the pupils he taught when clinical professor in University College Hospital, the usefulness of the thermometer in recognizing febrile diseases does not seem to have been hitherto sufficiently appreciated in the medical schools of this country.

## SECTION II.—INFLAMMATION.

**Definition.**—*A complex morbid process characterized,—(1.) By a suspension of the concurrent exercise of function among the minute elements of the tissue involved; (2.) By stagnation of the blood and abnormal adhesiveness of the blood-discs in the capillary vessels contiguous to the tissue-elements whose functions are suspended; (3.) By contraction of the minute arteries leading to the capillaries of the affected part, with subsequent dilatation and paralysis of the contractile tissue of the affected bloodvessels. The nutritive changes between the blood and the minute component elements of the affected tissue become visibly altered, and although an appreciable interstitial exudation does not necessarily follow, yet a constant tendency betrays itself to the occurrence of an interstitial exudation, but which, under proper regimen and proper remedies, is often abortive. When an exudation follows as a result of the inflammatory state, it is apt to be associated with an unhealthy condition of the blood, and of the blood-plasma, and to be associated with varied forms of new growth, according to,—(1.) The elementary structure in which it*

occurs; (2.) *The special zymotic, constitutional, or local disease with which this complex morbid process may coexist; and (3.) According to the progress of the inflammation, the amount and suddenness of the effusion, the extent of tissue involved, the diminished vascularity, and the powers of absorption of the surrounding parts.*

**Pathology.**—As it is not possible clearly to define the limits of natural processes, it is not possible to give a correct definition of inflammation. It is a process the most important of all morbid states; and a knowledge of its phenomena, the laws which regulate its course, and the relations which its several events bear to each other, have been always considered as “the keystone to medical and surgical science,” and the “pivot upon which the medical philosophy of the time has revolved.”

It is not wonderful, therefore, that much has been written on this subject, more especially since microscopic research has been brought to aid in the investigation. Among the many who have investigated this morbid process with success, and by whose original observations its study may be said to have begun, the names of Wilson Philips, John Thomson, Gendrin, Kaltenbrunner, Gerber, and Müller; and more recently those of Alison, Lebert, Gulliver, Addison, C. J. B. Williams, Bennett, Wharton Jones, Henle, Virchow, Paget, John Simon, and Joseph Lister, are well known; and no account of inflammation can be complete which does not embrace the results of the labors of these men.

The early experiments which illustrate the nature and phenomena of inflammation, have been made chiefly on the web of the frog's foot, as well as on the folds of the frog's mesentery; and the phenomena are found to correspond in all essential points, with the results of experiments performed on the more or less transparent parts of warm-blooded animals, such, for example, as the wings and ears of bats, the ears of rabbits, the mesenteries of these animals, and the brains of rabbits and of pigeons. As a general result of such experiments and observations, it may be stated, that the chief constituents of the inflammation-process, are to be found in altered conditions of the healthy nutritive changes—the phenomena of the abnormal state becoming more or less obvious by the redness, swelling, heat, pain, impairment of function on a large scale, and sometimes exudation in the part affected.

**Phenomena and Theory of the Inflammatory Process.**—The process is one in which many stages of morbid action are passed through, and which reaches its acmé when the serum of the blood and the *liquor sanguinis* transude through the walls of the bloodvessels of the inflamed part, without rupture, into the surrounding texture. This has been termed “exudation.”

The series of complex changes through which the inflammatory process is seen to proceed, as observed in the transparent parts of animals under the microscope, are found to occur nearly in the following order: 1st. The beautiful experiments and observations of Mr. Joseph Lister, Professor of Surgery in the University of Glasgow, clearly prove that a suspension of the concurrent exercise of function among the minute elements of the tissue involved, is the

primary lesion in the congestion of inflammation, and which immediately leads to—2d. Inflammatory derangement of the blood, which, in the vicinity of the impaired tissue-elements, tends to assume the same characters as blood always assumes when it is in contact with ordinary solid matter, and which renders it unfit for transmission through the bloodvessels. But a return of the tissue-elements to their usually active state, will be associated with a restoration of the blood to the healthy characters which adapt it for circulation (*Royal Society, June 18, 1857*). 3d. The arteries of the affected part are narrowed, and the blood flows through them with greater rapidity. 4th. The same vessels subsequently become enlarged, and the current of blood is slower, although uniform. 5th. The flow of blood becomes irregular. 6th. All motion of the blood ultimately ceases, and complete stagnation ensues. 7th, and lastly, The liquor sanguinis may be exuded through the walls of the bloodvessels, sometimes accompanied by the extravasation of blood-corpuscles, owing to rupture of the capillaries.

These different phenomena are associated with the production of the more obvious symptoms, namely, redness, pain, heat, and swelling. But although these changes are here mentioned consecutively, it is not to be understood that in every instance of inflammation, such changes can be traced in distinct succession. The changes are to be studied as *nearly concurrent*, rather than as a distinct series of events, of which each stands in the relation of a consequent to one or more of its antecedents; so that, starting from impaired function of the element of tissue, the stagnation of blood in the capillary vessels, we must observe the various stages in the process almost as concurrent phenomena, which, for the purposes of study, are here enumerated in sequence.

An analysis of these concurrent phenomena has shown that the conditions for the healthy nutrition of the part are materially changed, being somewhat as follows:

I. The supply of blood to the part is altered,—(1.) By the changes in the bloodvessels, especially the narrowing of the arteries, and subsequent enlargement of the capillaries; (2.) By the mode in which the blood moves through them.

The narrowing of the arteries, in the first instance, may be demonstrated under the microscope, by the application of warm water simply to the web of the frog's foot; and the same phenomena are presumed to occur in man, for the following reasons: Sudden operations of the mind, and the application of cold produce paleness of the skin—an effect which can only arise from contraction of the minute arteries, and the diminution of the quantity of blood thereby conveyed by them. The subsequent enlargement of the capillaries is presumed to be a constant event in the inflammation of a part. It usually extends to some distance around what may be considered as the chief seat, centre, or focus of diseased action, and in some textures the enlargement and reddening are confined to the vascular parts in the vicinity. To this condition of the blood and bloodvessels, is to be ascribed the usually first observable symptom of inflammation in a part, namely, the redness. But there are also

many circumstances under which inflammation has existed, and yet no redness is apparent in the part itself. Thus we often open the body of a patient that has died of phthisis, and find the intestine ulcerated; but, so far from being redder, it is paler than natural, and, so far from being thickened, it is thinner than usual. We often find the cartilages of the joints ulcerated, and yet not a trace of a red vessel. In cases of bronchitis, with purulent expectoration, if the lungs be washed, so as to remove the morbid product, the most experienced anatomist may be unable to determine whether the parts are in a state of health or disease. Take the arterial system, and how often do we find the aorta thickened and thinned, softened and indurated, ulcerated, and its elasticity entirely destroyed, and yet not a red vessel to be seen; and when the patient has neither complained of the slightest sensation of pain, nor of any feeling of heat in the part during life? A large abscess may form in the brain or areolar tissue, or pus may be effused into the cavity of the abdomen, without any appearance of redness, or even evidence of having been preceded by any suffering. Although in certain parts, as the cornea and the articular cartilages, the ulcerated intestine, or the bronchi, the arterial tissues, and the seats of abscesses, the previous existence of inflammatory action is obvious from the effects produced, and where no bloodvessels existed, obvious to the eye, assisted or not by the microscope, yet it is, for the most part, found that enlargement of the bloodvessels of the adjacent parts, and especially of those from which the diseased part derives its nutrient supply, is a constant phenomenon, purely functional, and which appears to be developed indirectly through the medium of the nervous system. In inflammation of the cornea, for instance, the bloodvessels of the sclerotic and conjunctivæ are enlarged. In ulceration of the articular cartilages, the surrounding synovial membrane, and the articular extremities of the bones, are more fully pervaded with enlarged bloodvessels. The *vasa vasorum* of the aorta, round the morbidly thickened part, are also the subject of enlargement, and the channel of increased supply of blood. There is, therefore, no doubt that the conditions favorable to the existence of redness are always present to a greater or less degree at the early period of inflammation; and whether the redness be always present, or only slightly perceptible, the same impairment of function among the minute elements of the tissue, and increased adhesiveness of the blood-discs, take part in the development of the inflammatory process.

The enlargement of the bloodvessels varies. It may be hardly perceptible, or it may increase their diameter to two or three times their natural size. John Hunter established this stage of the inflammatory process in the ear of a rabbit, by thawing it after it had been frozen: the rabbit was killed during the process, and the head being injected, the two ears were removed and dried. Wood-cuts representing the comparative conditions of the two ears may be seen in the first volume of Paget's *Surgical Pathology*, page 295. The bloodvessels of the inflamed ear became greatly larger than those of the healthy one, and it was found that arteries before invisible, in the

healthy state of the rabbit's ear, were brought clearly into view during the stage of the inflammatory process.

The redness of an inflamed part is of various intensity and shade, according to the degree of the inflammation, its stage, and the structure of the part affected. Its shades pass from a light rose-color to a deep crimson, or even purple. It assumes the form of points where congeries of minute bloodvessels are concerned; or streaks, as where the vessels of fibrous structures are inflamed, as in tendon; or a series of minute and fine ramifications, as in synovial structures; and generally it may be stated that the form of the redness derives its character from the normal arrangement of the capillaries of the part. The redness is most intense towards the centre of diseased action, gradually softening down towards the circumference, where the conditions of health exist. This gradual shading off serves to distinguish the redness of inflammation from the redness of extravasation. The margin of an extravasation is defined, its redness cannot be removed by pressure; while the disappearance of inflammatory redness under pressure is, to a certain extent, a measure of the activity of the circulation in the part. The brighter hues generally attend ordinary active inflammation;—the darker hues of inflammatory action are generally associated with some specific cause of disease, a feeble action of the parts, or a tendency to gangrene. The increased depth of color is mainly due, in the first instance, to the congestion and stagnation of blood in the existing vessels, and not in any measure to the formation of new ones. The redness, however, always appears more than proportionate to the enlargement of the bloodvessels; and we find that the red corpuscles are intensely adherent in the enlarged capillaries.

The dilated vessels of an inflamed part appear crammed with red corpuscles, which lie or move as if no fluid intervened between them, or as if they were imbedded in a hyaline substance due to the solidification of the fibrine of the liquor sanguinis. An increase of redness is sometimes seen to depend upon extravasation of blood, or the effusion of the coloring matter of the blood-corpuscles, as well into the spaces between the blood-corpuscles as into the adjacent tissue through the walls of the bloodvessels. Lastly, the redness is sometimes intensified (as HUNTER first suggested, and microscopic examination subsequently proved) by the passage of the blood, unchanged, from the arteries into the veins. No new formation of bloodvessels is necessarily concerned in the redness of inflamed parts. It is only when inflammation has subsided that new vessels are formed, and pass into any new growth of tissue which may have arisen, as if for its nutrition, development, and continued growth, or to effect its subsequent removal, degeneration, decay, or absorption.

Peculiar changes of shape are associated with enlargement of the bloodvessels, consisting chiefly of tortuosity of distribution and aneurismal or varicose dilatation. The aneurismal or varicose state is seen to take place most frequently in the soft textures, as in the brain, where it is a frequent condition of the inflammatory red softening (KÖLLIKER and HASSE); and in subcutaneous tissue, the points



of what appears to be extravasated blood are aneurismal dilatations of capillary vessels filled with the red corpuscles (LEBERT).

These varied conditions of the bloodvessels affect the motion of the fluid in the part, and consequently the supply of blood for the purposes of nutrition. Generally it may be stated that there is stagnation of the blood in the focus or centre of severe inflammation; and this stagnation is surrounded by a state of fulness of vessels and slow movement of the blood, while around, and more distant still, there is fulness of the vessels, with a rapid movement of the blood. From the discrepancy existing among observers regarding the statement as to whether the motion of the blood is slower or quicker when the vessels are contracted or dilated, there is evidence that the contraction alone of a vessel, or its dilatation alone, is not always sufficient to cause the current of blood to be either slow or quick. Other conditions are at work which contribute in no small degree to accelerate or slow the rate of movement in the vessels. Besides the force of the heart's action, there is a mutual relation which subsists between the blood and bloodvessels and surrounding tissue, which materially influences the motion of the blood. In the healthy body this mutual functional relation between the minute elements of tissue and the blood is necessary to maintain it in a state fit for transmission through the vessels. The mere contraction of the arteries leading to a part does not tend to stagnation of the blood in the capillaries of the inflamed part; on the contrary, the movement onwards of the blood in the vessels is influenced or modified by the vital functional processes going on between the capillary vessels and the surrounding elements of tissue, and which has been variously named the "capillary force," the "vital force," the "nutritive force;" it is also mainly influenced by the action of the heart itself, and by the physical condition of the vascular tubes through which it has to pass. Accordingly, at first, with contraction of vessels, the current has been described as being quickened. It also sometimes slackens, or even retrogrades for a time, and not unfrequently oscillatory movements may be noticed. But when dilatation is complete, the blood flows with rapidity, and a greater quantity passes during a given time than in the unexcited state of the parts. This is known as the state of "determination of blood to a part," or "active congestion." The natural function of the part thus becomes simply exalted; and it may be said that a step beyond this will pass the confines of that neutral ground which exists between health and disease. With an increased circulation, and such "determination of blood to a part," functional activity is not only maintained, but is promoted and increased; and unusual transudation of the nutrient material may take place, chiefly of the serum of the blood. Hence the œdema which surrounds an inflamed part. After a time the motion of the blood becomes slower, while the volume propelled is increased, and the retardation gradually increases till the blood-corpuscles are no longer propelled, floating in their *liquor sanguinis*; but accumulating in masses, they advance by a jerking intermittent motion, till at last complete stagnation takes place. The blood-corpuscles now

detained exhibit a marked tendency to adhere alike to the walls of the vessels and to each other; thus accumulating together and sticking in the capillaries, while the *liquor sanguinis* flows onwards. To this condition the term "stasis" has been applied. In the immediate neighborhood, and surrounding the part which is in the condition of *stasis*, the circulation of the blood goes on with increased rapidity: it may even pulsate in the arteries and oscillate in the veins, while it moves with a uniform but rapid flow through highly distended but less turgid vessels. When these conditions exist simultaneously, and the true morbid process is completely established, the capillary vessels may burst, causing hemorrhage or extravasation into the surrounding tissue, or the serum and *liquor sanguinis* may transude through their walls, without rupture, into the surrounding texture.

The "determination of blood to a part" here noticed, characterized by dilatation of the arteries with increased flow of blood through the capillaries, must be distinguished from the "congestion of inflammation," characterized by the accumulation and stagnation of red and white corpuscles in the vessels, tending to be abnormally adherent to each other and to the vessels. Both of these phenomena, namely, "determination" and "congestion," may result from irritation. The dilatation of the arteries seems to be immediately developed through the medium of the nervous system, while the accumulation of the blood-discs and stagnation of the blood is the immediate and direct result of impaired or suspended function of the minute tissue-elements contiguous to the capillary vessels.

The "determination of blood" and dilatation of the arteries lead to no change in the quality of the blood itself; on the other hand, accumulation and stagnation of blood, in the congestion of an inflamed part, are associated with increased adhesiveness of the red and white discs. Mere determination of blood becomes obliterated after death by the post-mortem contraction of the arteries, whereas the congestion of inflammation is persistent. It is an evidence of organic lesion declaring itself as distinctly in the dead as in the living; and thus the most important, if not the only sign of the early stage of inflammation having occurred during life is recognizable, on dissection, by the intense redness due to the accumulation of red discs adherent to each other in the minutest ramifications of the vessels, and not due to distension of the vessels merely.

Such is a statement of the facts ascertained regarding the early phenomena of the inflammatory process; and they are of such a kind that, with the facilities of study which ought now to be within the reach of every student of medicine, he ought to make such experiments as have been already noticed, or see them made by others, and thus really appreciate the steps of that morbid process which he requires to treat so extensively in practice, and of which he can form but a faint conception from the most lucid description.

II. The constitution of the blood is altered as regards its adaptability to nourish the part.

The nature of this alteration cannot be chemically expressed; but microscopical observation has established a fundamental fact, namely,



that the tissues through which the blood flows have such special relations to the living fluid that, in the healthy state, the functional activity of the minute tissue-elements maintains the blood in a state fit for transmission through the bloodvessels; and the first change observed in the blood, subsequent to any impairment of function of tissue-elements, is an increase of adhesiveness of the red as well as of the white corpuscles: but the white corpuscles are now known to be susceptible of much greater adhesiveness than the red; so that slight irritation, leading to impairment of function, causes stagnation of the white sooner than of the red discs. The blood is not thus altered in the first instance throughout its whole mass; but the change is a local one, confined to the seat of the inflammatory process. At one time it was believed that the blood was altered in its constitution chiefly by an increase of the fibrine and the white corpuscles;\* but it is now found that the white or rudimental corpuscles of the blood cannot be separated from the fibrine by any known process; consequently the relative amount of fibrine cannot be correctly stated in relation to the blood. And, as in many inflammations these corpuscles are increased, as well as in many conditions, such as pregnancy, in which no inflammatory process exists, the blood is similarly altered, it is not known how much of change is due to fibrine or how much to the white corpuscles. The generation and accumulation of large numbers of white corpuscles in the vessels of an inflamed part is not now received as a fact. The phenomenon may be true as regards some frogs, but not as regards warm-blooded animals; and it is consistent with the experience of three most eminent pathologists who have experimentally examined this subject—namely, Mr. Wharton Jones, Dr. Hughes Bennett, and Mr. Paget—that an especial abundance of white corpuscles in the vessels of an inflamed part is neither a constant nor even a frequent occurrence. Dr. Hughes Bennett's researches, relative to leucocythæmia, have shown that even the most extreme abundance of white corpuscles in the blood has no tendency either to produce or to aggravate inflammations.

A remarkable phenomenon presented by the red blood-corpuscles in inflammation was first observed in 1827 by Mr. Lister, Sr., and by Dr. Hodgkin, and afterwards accurately described by Mr. Wharton Jones.

They observed that when healthy blood is received on a glass plate, or the clean surface of a polished lancet, and immediately examined, the corpuscles lie diffused in the *liquor sanguinis*, but in about half a minute they run together into piles or rouleaux, which arrange themselves in small meshed networks. But if a drop of blood from a patient with acute rheumatism, or with an inflammation, be similarly examined, piles of red corpuscles instantly form,

---

\* Andral and Gavarret showed that the proportion of fibrine in the blood was augmented in inflammations, when sufficiently severe or extensive to affect the system. In health the average proportion is three parts in 1000, and in cases of severe inflammations it has been found to rise as high as eight, nine, or ten parts in 1000. This increase commences as soon as the inflammation is established, and ceases when the process begins to decline.

and are clustered into masses, leaving a network with wide interspaces.

This appearance of itself, however, is not a sure sign of inflammation. It may be observed in the blood of the chlorotic female as well as in the pregnant one; in those also in whom a plethoric condition as regards the blood exists; in persons in health, whose circulation has been much accelerated, as by violent exercise; and it appears to be the natural state of the blood of horses. It is a phenomenon resulting from an increased tendency to aggregation of the blood-corpuscles, and gives a granular appearance to a thin layer of blood when viewed with the naked eye. When blood is drawn off in quantity, the phenomenon is associated with the formation of what is termed the "buffy coat," as the clustered blood-corpuscles, rapidly sinking, subside to some distance below the surface before the fibrine and the white corpuscles begin to coagulate.

However indefinite and uncertain the changes may be, as observed upon a small portion of the blood, it cannot be doubted that the blood stagnant or retarded in an inflamed part undergoes important alterations; and by a constant succession of such changes the whole fluid may come at length to be materially altered, as indicated by the general effects and constitutional disturbance, extending throughout the nervous and the vascular system, and which may ensue in the train of an inflammation of purely local origin. It is probable that local changes ensue in the blood similar to those we shall have to notice as taking place in the products of growth in and amongst the elements of tissue during the inflammatory process. There is no doubt, as Wharton Jones has shown, that fibrinous coagula occasionally form, and even degenerate, within the bloodvessels. When the stagnation of the blood is not constant, these fibrinous coagula are carried away into the general circulation, giving rise to the phenomena of embolism (to be afterwards described) in the capillary vessels of some of the more solid viscera, such as the brain, lungs, liver, spleen, or kidneys. By the degeneration of such coagula the whole mass of blood may be infected, and constitutional disturbance excited, producing sometimes various and wide-spreading suppuration,—as when purulent infection is consequent on local injury, or when a blood-clot passes upwards, and becoming lodged in the cerebral vessels, induces the state known as softening of the brain.

There are many points or questions deserving of attention regarding the theory of the inflammatory process; but it is also obvious that in a handbook such as this, any mere analysis of speculative doctrines ought not to take up much space. The following statement will therefore merely embrace as much as possible of those topics of special interest which a more extended and accurate physiological knowledge of the process of inflammation has shown to be the proper objects of more extended inquiry.

In the first place, as to the primary seat of the inflammatory process, there can be little doubt, from the phenomena already described, as well as from the results of dissection, which show the progress and effects of the process, and from the experimental

researches of Hunter, Thomson, Wilson Philips, Hughes Bennett, Wharton Jones, John Simon, Paget, Lister, and other observers, that the vital morbid process known as "inflammation" is connected with the minute capillaries and the most minute elements of tissues which they nourish. Questions relative to the theory of the process are therefore found to be intimately connected with the histological and physiological relations of these parts.

During the earliest period of the process—the period of increment, or of incubation, as it has been termed—it appears to be the inherent properties of the minute component elements of tissues which first undergo a change, and combined with the reflex actions of the nervous system, seem to maintain, to promote, or to increase the activity of the subsequent stages.

The simplest effects upon the minute elements of tissue, and upon the bloodvessels, are seen to follow the application of the mildest or slightest physical or chemical agents, but which, operating powerfully, are also capable of extinguishing altogether the life of these elements of tissue. When the action induced is mild and gentle, the tissues become incapable of performing their wonted functions; and, provided the mechanical or chemical agency has not been too severe, the impairment of function may subside, and the tissues will return to their normal state of functional activity. This is "resolution" of the inflammation.

Such irritant causes acting either immediately from without, or through the blood, or through the instrumentality of the nerves, each component texture of the part becomes affected as soon as it is brought in contact with the irritant. A gradual contraction of the arteries takes place—the contraction following at some interval after the application of the stimulus—is slowly accomplished, and persists for a variable length of time. Relaxation then no less gradually ensues, when the capillaries open up and slowly dilate till they acquire a size larger than they had previous to the application of the stimulus.

The minute arteries have been shown by the histologist to possess in abundance the structural elements of the non-striated contractile tissue; and in this respect they closely resemble the constitution of the muscular fibre of the intestine. Accordingly, the contractions they undergo have been considered as analogous to *spasms* (as Cullen first suggested); while the succeeding dilatation may be of the nature of *relaxation*, and ultimately of *paralysis*. This paralyzed state is shown from the fact that the same vessels now dilated will not contract upon a re-application of the same stimulus which before made them contract. If the stimulus is made with a needle upon the vessels in the transparent parts of an animal, the needle may be repeatedly drawn over such dilated vessels and no contraction will follow; but with a stronger stimulus, such as that of heat, they may be made to contract again, and even close; and this state of contraction may persist for a whole day, before the vessels again open up and permit the blood to flow (PAGET). On the other hand, the true capillaries seem totally destitute of any structure known to be contractile. They merely consist of a delicate homogeneous

membrane, beset with occasional nuclei. A film of collodion is not more homogeneous nor more continuous than the membrane of a capillary (VIRCHOW). Whereas the minute arteries (some of them less even in calibre than capillaries) possess distinct coats, one of them consisting of a single layer of muscular (or contractile) fibre-cells, wound spirally round the internal membrane of the blood-vessel, so as to encircle it from one and a half to two and a half times. The arteries, to their smallest branches, are sometimes contracted to absolute closure, and at other times are widely dilated; whereas the capillaries are never entirely closed, nor do they present any variations in diameter which are not due to the elasticity of their parietes (LISTER, l. c.).

The most interesting point in the whole process is perhaps that which embraces an inquiry into the cause of the "stasis," or stoppage of the blood, and the exudation of the *liquor sanguinis*, which are the most difficult phenomena to explain consistently with physiology. This is a point which I think the observations of Professor Lister have so very beautifully illustrated; but the explanations of other eminent pathologists and experimentalists, if not universally satisfactory, serve to present the subject in a variety of aspects to the mind, which cannot fail to be both interesting and practically instructive. Henle, Simon, Bennett, Williams, Rokitsansky, and Paget, have all helped to elucidate the process by the following theories:

The theory of Henle, or, as it is sometimes called, the "neuro-pathological theory," assumes that the stimulus, acting on the sensory nerves of the part, excites in them a state which, being communicated to the spinal nervous centre, is reflected on the vascular nerves, occasions their paralysis, and therewith paralysis also of the contractile coat of the bloodvessels. Various modifications have been made upon this theory; but as the phenomena have been seen to take place in the case of absence of a spinal cord, and in division of the roots of the nerves, and in section of the lumbar and sciatic nerves, such facts are subversive of the hypothesis. Henle considers the stasis as a necessary physical consequence of this dilatation of the bloodvessels, and this stasis, together with the relaxed and dilated state of the vessels, favors the exudation of serum, the consequence of which is, that the plasma of the blood in the part becomes inspissated by a preponderance of albuminoid matter over the salts. This inspissation of the plasma determines endosmotic changes in the red corpuscles, in consequence of which they are disposed to aggregate.

Simon propounds the view that the phenomena are due, not to a reflex action, but to a direct change effected by the living molecular structure of the part on the blood which traverses it, or on the vessels which convey that blood.

Bennett ascribes the change as due to a vital force actively operating through the tissues which lie outside the vessels, and which is the only active agency causing the approach of the colored particles to the capillary walls of the bloodvessels, and the passage through them of exudation.

Paget supposes a mutual relation to exist between the blood, its vessels, and the parts around, which being natural, permits the most easy transit of the blood, but being disturbed, increases the hindrances to its passage.

Dr. C. J. B. Williams considers that an essential part of inflammation is the production of numerous white globules in the inflamed vessels, and that the obstruction of these vessels is mainly due to the adhesive properties of these globules.

Rokitansky is of opinion that the condition of *stasis* proceeds,—1st. From the sticking together of the blood-corpuscles, the heaping up and wedging of them in the capillaries, while the plasma in part flows off towards the veins; 2d. From the inspissation of the plasma, occasioned by the exudation of serum through the dilated and attenuated walls of the vessels, and its saturation with fibrine and albumen; 3d. From the heaping up of the colorless corpuscles, *i. e.*, the nucleus and cell-formations, together with blood-globules; from their sticking together, and from the delicate hyaline, fibrinous coagula which develop themselves among them. Rokitansky considers this to be the most important moment in the inflammatory process, since on the one hand it very specially throws light upon the phenomena of *stasis*, and on the other hand it comprehends the plastic processes which take place in the heaped-up and stagnant blood. It separates in this way the process of inflammation from a merely simple one of exudation. The elementary formations above-mentioned are not merely swept together towards the place of *stasis*, but they originate as new formations in the stagnant blood, which generally presents remarkable alterations.

Wharton Jones describes the progress of *stasis* as consisting,—1st. Of the adhesion of collapsed and dark-red blood-corpuscles to the walls of the vessels; and 2d. The adhesion of other blood-cells to these. The first adhesion of the blood-cells usually takes place at a bifurcation, and the stagnation of blood is seen to begin in those capillaries which are least in the direct course from the artery to the vein, depending in a great measure upon the inspissation of the plasma, or its increased quantity of fibrine and albumen.

Whatever explanation may be given or accepted as to how the phenomena of inflammation in a part are brought about, our views regarding the essential nature of the process have been hitherto modified according as this complex morbid state has been studied by its effects as seen on the dead rather than on the living body. There are some especially eminent pathologists whose combined observations have of late done much to convey a clear notion of the essential nature of this complex process, namely, Alison, Virchow, Bennett, Simon, Goodsir, Redfern, and Lister. While Dr. Bennett regards an exudation from the bloodvessels as the necessary constituent of inflammation, Alison and Virchow, on the other hand, recognize the morphological changes of the living tissues, such as have been described in inflammation, as betraying merely a *tendency* in a part to such a local change as exudation amongst its structure. That *local tendency* may be so slight that hardly any difference can be appreciated between the healthy changes attendant on normal



nutrition, and those changes between the blood and the minute tissues which are of such a kind that a morbid change (inflammation) is established in the elementary components of the tissues themselves, without any appreciable exudation having taken place either amongst the interstices or upon the free surfaces of membranes. To such a condition Virchow gives the name of *parenchymatous inflammation*, meaning thereby that it is a process established locally between the capillaries, the blood, and the component elements of tissue, and expressed by a tendency merely to the effusion from the bloodvessels of such plastic material as may eventually take place.

*Inflammation* may thus exist as a local morbid process, characterized by an abnormal condition of the nutritive changes between the capillaries, the blood, and the component elements of a texture, without any appreciable exudation. Such an abnormal condition will, under proper regimen and proper remedies, in a case of simple inflammation, seen from the first, completely subside, no interstitial exudation ever taking place.

Examples of this simple form of inflammation have been fully illustrated by Goodsir and Redfern in this country, by their demonstrations of what takes place within the large cells of cartilage. The cells become larger, the number of nuclei increases, and some, or all of them, may undergo fatty metamorphosis under the influence of this the simplest form of inflammation, and which is only manifested by this abnormal nutritive process between the blood and the cells, and which at once leads to these changes *within* the cell-elements of tissue, described by Virchow as a "cloudy swelling" of these parts, *e.g.*, the cells of the uriniferous tubes, and those of the mucous membrane in the state of catarrh. In this abnormal nutritive process, however, there is a constant tendency to the interstitial exudation of a hyaline material, which may become fibrous or filamentous, and ultimately soft and gelatinous. Virchow, Weber of Bonn, and His, have demonstrated similar changes in the cells of the cornea.

Thus the minute and penetrating observations of Virchow have given a more comprehensive meaning to the process of exudation than it has hitherto, in this country, been understood to signify; and such alterations as he and others have described in the elements of the tissues of an inflamed part have been in a great measure overlooked, except by Dr. Alison and Mr. Simon. The latter especially states that the irritation of the inflammatory process is independent of the nervous influence, *but is a direct change operated by the living molecular structure of the part on the blood which traverses it, or on the vessels which convey that blood.* Dr. Alison, also, long recognized the *tendency* to interstitial exudation as attending such vital changes in the constituent elements of a part, and which entitled it to be considered inflamed. The accurate observations of Virchow, Goodsir, and Redfern have shown that such primitive changes do take place before those more palpable phenomena occur which constitute the exudation as described by Bennett, namely, the exudation of decolorized lymph into the interstices between the constituent elements

of a texture. Both sets of phenomena alike show that *inflammation is only one of the various shades of deviation from the normal process of nutrition,—a diseased action tending to a local lesion* (*British and Foreign Medico-Chirurgical Review*, January, 1854). That the irritation of inflammation is in some measure independent of the nerves, the following interesting experiment, related and performed by Mr. Simon, may be quoted in proof:

“A patient had complete anæsthesia of the fifth nerve, dependent (as a post-mortem examination subsequently showed) on its organic disease; the conjunctiva, as well as the integument of the face, was utterly insensible: not only was the function of the nerve destroyed, but those reflective nutritive changes of which I have already spoken had taken place, and had exhausted themselves; showing that the nerve was spoiled for participation in the acts of nutrition (whatever they may be) no less than for its more obvious uses as a medium of conscious sensation; the cornea had undergone ulceration, and had healed again. The following experiment was carefully made: The lids being held open, a single granule of cayenne pepper was laid upon the insensible conjunctiva; in a few moments it had become the centre of a very distinct circle of increased vascularity, the redness of which slowly became more and more distinct as long as the stimulus was suffered to remain, so that, in its removal, there was a very evident circumscribed erythema on the surface of the membrane. I considered myself justified in believing that this change occurred without any intermediate nervous excitement; not only because the history of the case would lead me to consider the fifth as annihilated; not only because the experiment was totally unattended with sensation; but likewise because there was the very remarkable absence of that sympathetic phenomenon which the faintest remnant of nervous excitability would have produced—namely, there was not the slightest trace of lachrymation” (*Lectures on General Pathology*, p. 76).

Further evidence might be submitted from the papers of Mr. Joseph Lister to the Royal Society, already referred to.

Such being the essential nature of inflammation, it is easy to understand how reasonable is that doctrine which teaches “that the process of inflammation is susceptible, at all times and in all countries, of very great variety as to extent or intensity, and especially as to the constitutional affection associated with it or consequent upon it.”

**Products, Effects, or Events of Inflammation.**—Care must be taken not to put the *products* of inflammation in place of the *symptoms* of inflammation. When the local impairment of function of the minute elements of tissue in process of inflammation is *confined to a small space*, or is *carried on upon a minute scale*, or *rapidly abates*, the inflammation is said to *terminate by resolution* as a general principle; that is, the abnormal action ceases, interstitial exudation does not take place, the tendency to further impairment of function is subdued and passes off, and the part is left apparently as it was before. If, however, interstitial exudation has taken place, and *resolution* is to be effected, the return of the part to health may be followed, for some time, by some impairment of its structure and function.

After the process has thus gone a certain length, an increased local growth of cells, and their liquefaction or reduction to a state capable

of absorption (what Dr. Addison calls *cell-therapeutics*), are essential to the restoration of the part. Before the process has attained such a length, however, *resolution* may be simply effected by a gradual return of all the parts to a natural state: a mere retracing of the steps by which the natural actions had been departed from sufficiently describes the process (PAGET).

The process of *resolution* has been closely watched by Mr. Paget. He has seen, in those cases where impairment of function and actual lesion had taken place, that fragments of fibrine, washed from the blood in the vessels of the injured parts, were borne along and floated in distant vessels. The observations of Dr. Kirkes, also, leave no doubt that similar changes may occur in warm-blooded animals, and may be the source of great evils; may be, indeed, productive of some of those constitutional effects yet to be noticed, by carrying the materials of diseased or degenerate blood from a diseased organ to one that was previously healthy. When the disappearance of the inflammation is unusually sudden and rapid, the event is technically called "*delitescence*," and if at the same time the symptoms of inflammation appear at another part not anatomically connected with the part first diseased, the event is called a "*metastasis*."

When the process does not confine itself to the simple expression of altered nutritive changes between the constituent tissues of a part and the blood; but when the tendency to exudation amongst the interstices of texture continues, and does not subside, as already explained,—namely, by *resolution*,—then it is that (1) such a material is separated from the blood as will become a medium or *nidus-substance*, in which many changes connected with the growth of new particles, granules, or cell-forms will take place, and the phenomena of which have been so well described by Bennett, Gluge, Paget, Virchow, Beale, and John Simon; and (2) coincident with this exudation, and the changes which it undergoes, the tissue of the part itself sustains serious alterations. For in all such inflammations especially of the more vascular parts, when there is increased exudation from the bloodvessels, there is a great deterioration of the surrounding elements of tissue. The texture is rendered soft and easily torn, and by such changes of cohesion the elasticity of parts (a circumstance often of very primary importance) becomes greatly altered and impaired.

These changes, therefore, Mr. Paget happily describes as consisting of,—(1.) *Productive* effects—that is, effects resulting from the growth of new particles, granules, or cell-forms, from pre-existing germinal elements of tissue, and which are susceptible of further development, and also of *degeneration*; (2.) *Destructive* effects, such as *softening*, *degeneration*, *absorption*, *ulceration*, and *death* of tissue.

**Productive Effects of Inflammation—Inflammatory Effusions or Exudations.**—These consist of,—1. Serum; 2. Blood; 3. Fibrine; and 4. Mucin. These last two are the only *true inflammatory exudations*.

1. *Serous Effusions*.—The effusion of pure serum is said to be very rare. In inflammation of a serous membrane, as the pleura, the fluid effused is not only greater in quantity than natural, but it is also greatly altered in quality. In health the serous secretions





cient to cause the exudation of fibrine, independently of obstruction to the circulation; and the cause of the greatest differences in the nature of exudations, is to be found in the special constitution of the irritated parts (VIRCHOW).

It has been clearly shown (SIMON, LEHMANN, BEALE), that there are two essential characteristics of inflammatory effusion,—(1.) It tends to contain certain ingredients in larger proportion than that in which they exist in the blood—excess of chloride of sodium and of phosphates and albumen; (2.) Organic forms find in it a suitable place for growth.

The site of effusion resulting from inflammation, is important, as sometimes constituting the chief element of danger—a danger sometimes immediate, from the mechanism of the parts affected. A large quantity of fluid is often poured out in a very short time. The cavity of the pleura may fill in a few hours, and the lung may be compressed by it to a half or third of its bulk: and if both pleural cavities become thus affected, constituting *double pleurisy*, the patient must die from suffocation, if not at once relieved, by allowing free vent to the fluid. This operation is called *tapping the chest*, or, technically, "*paracentesis thoracis*." Serous effusion into the areolar submucous tissue of the glottis may also produce almost immediate death from suffocation, unless the cavity of the larynx is immediately opened to admit the air to the lungs (WATSON).

2. *Blood Effusions or Extravasations*.—Such chiefly occur from rupture of the new vessels developed in the newly formed material, which has just become vascular (ROKITANSKY). In the ordinary course of internal inflammations, extravasations of blood are rare, and betoken an unfavorable state of the constitution generally, such as occurs in typhus fevers, in scurvy, in purpura, or in syphilis. The post-mortem evidence of such extravasations is the presence of a colored cicatrix at the spot of rupture and effusion, and the color is found to be due to the presence of hæmatoidine, generally in the form of a mass of aggregate crystals, composed of minute rhombic columns, and which may be considered as the regular typical ultimate form into which hæmatine is converted in any part of the body where considerable masses of extravasated blood continue to lie for any length of time, *e. g.*, apoplectic clots and coagula in the Graëfian vesicle of the ovum after menstruation (VIRCHOW).

Mr. Paget correctly observes that we must not confound with hemorrhages the cases in which the inflammatory products are merely blood-stained, *i. e.*, have acquired a more or less deep tinge of blood, through the oozing of some of its dissolved coloring matter. The natural color of inflammatory new formations is grayish or yellowish-white, and even when they have become vascular, their opacity in the recent state prevents their having any uniform tint of redness visible to the naked eye. When inflammatory products present the tinge of redness, it is either because of hemorrhage into them, or because they have imbibed the dissolved coloring matter of the blood; and when this imbibition happens during life, or soon after death, it is important, as implying a cachectic, ill-maintained



Rokitansky describes these typical forms by the terms "*fibrinous*" and "*croupous*," and Dr. C. J. B. Williams by the names of "*plastic*" and "*aplastic*." Examples of each variety may illustrate the application of the terms. To the *fibrinous* or *plastic* variety belong the serous effusions already referred to, and perhaps also the granular, molecular, or fibrillated growths. The *corpuscular*, *croupous*, or *aplastic* forms of lymph are represented by those growths which never become consolidated, as in the early formed contents of vesicles in *vaccinia* and *herpes*; in the *fluid of blisters* raised in cachectic patients; in some instances of *pneumonia*; and in some forms of inflammation of *serous membranes*. In by far the larger number of inflammatory products these typical forms are mixed in various proportions; and the larger the proportion of corpuscles in new growth, the greater is the probability of suppuration, or of some other degenerative process, and the more tardy is any process of development into tissue, such as that of adhesions, indurations, and the like. In other words, the preponderance of granules, molecules, and fibrillated material in the new growth, is generally characteristic of the "adhesive inflammations;" the preponderance of corpuscles, or their sole existence in a liquid medium, is a general feature of the "suppurative inflammation." The hardness of inflamed parts is due to the former of these typical forms of inflammation, and is exemplified in the case of a *phlegmon* or *boil* before it suppurates; as also in a lung in a state of *hepatization*, when its textures are enclosed by lymph, "just as the stones of a wall are by the cement;" also in the hardening of a chancre.

On the surfaces of inflamed membranes the new growth forms a layer of a membranous firmness or consistence, to which the name of *false*, *adventitious*, or *pseudo-membrane* has been given. By this new growth the naturally opposed surfaces of parts which are inflamed are apt to adhere. This is commonly seen to be the case between such serous surfaces as the *pleuræ*, the *pericardium*, the *peritoneum*, or the *edges of a wound*. The inflammation associated with this organization is sometimes called "*adhesive inflammation*;" and Dr. John Thomson ascertained that this growth and organization might be effected between the surfaces of wounds in less than *four* hours after they were inflicted.

On the surfaces of mucous membranes may generally be seen the "*corpuscular*" typical form of new growth as a result of inflammation. It has little tendency to cohere, but grows in films, gelatinous masses, shreds, patches, or delicate casts of the surface upon which it was formed. The new growths in chronic catarrh of the intestines are an example; so are the membranes sometimes passed from the cavity of the uterus, and called *dysmenorrheal membranes*. In the "adhesive" form of inflammation the new growth of granules or of molecules may ultimately assume the form of *fibrous* tissue interstitial to the textural elements inflamed. Examples of this organization are seen in the laminated and nodular thickening of the capsules of the spleen, the thickening and induration of the periosteum, or the capsule of the hip joint in chronic arthritis; and by virtue of the peculiar tendency to contraction which fibrinous prod-



in the lymph deposited over a compressed lung, associated with empyema or hydrothorax.

2. The fibrine of lymph may undergo changes similar to what is known as fatty degeneration,—changes similar to those which occur in the *primordial lymph-cell* when it is transformed into pus. The two changes generally go on together. To the former change, namely, the fatty-like degeneration of the fibrine, Mr. Paget gives the name of "*liquefactive degeneration*:" the solid fibrine of inflammatory lymph that becomes again liquid when suppuration takes place, as may be observed in a hard mass of inflamed texture when it becomes soft.

This is a degeneration which brings the new growth into a state favorable for its absorption, or to the *resolution* of an inflammation. Examples of such an absorption may be seen in *rheumatic iritis*, and the observations of Dr. Kirkes on the rarity of adhesions of the pericardium in comparison with the frequency of pericarditis may also be explained in this way.

3. Melanic degeneration of lymph and new growths is not unfrequent, as in *peritonitis*.

Concurrent with these degenerations of the lymph-granules and molecules are the degenerations of the *corpuscular elements*.

1. They may wither, as in the dried-up pus of chronic abscesses.

2. The fatty degeneration of cells is said to be shown in their transition to the *granule-cell*, known also as the *inflammatory globule* of Gluge, or the *exudation-corpuscle* of Bennett. The history of the formation or growth of these corpuscles is still doubtful.

The description of them, as originally given by Gluge, in describing the alterations of blood in inflamed parts, is as follows:

He observes "that the blood-globules lose their tegument and their color. Their inner substance alone remains, which, however, does not remain solitary; but by means of a whitish connecting material the masses become agglomerated, and form dense, opaque, round groups, containing on an average from twenty to thirty of the smaller bodies, which examined singly, are quite light and transparent. By means of pressure or acetic acid the associated granules break down into the individual bodies, and we see that the opacity is merely owing to the association. The associated bodies have a diameter in the mass of from  $\frac{1}{80}$ th to  $\frac{1}{30}$ th of a millimetre; the single granules are from  $\frac{1}{800}$ th to  $\frac{1}{400}$ th of a millimetre. These associated bodies," says Gluge, "I have seen in the bloodvessels, so that we have not here to do with a fluid which, transuding through the coats of the bloodvessels, is changed into granules. They escape by bursting the capillaries."

That this cell or corpuscle is formed within as well as without the bloodvessels, is apparent from an examination of inflammatory lungs or brain-substance. The corpuscles may be seen to coat the bloodvessels exteriorly and interiorly to their walls; and the formation of the corpuscle of Gluge can also be traced through stages of development, as described by Vogel, Bennett, Kölliker, Hasse, and myself; as well as through stages of degeneration from the normal state of some corpuscular elements (textural or morbid), the occurrence of



are often found in the fibrinous clots of veins;—with their occurrence in the mammary secretion, in the softened parts of encephaloid cancer, in the vicinity of apoplectic effusions, and that generally they are extremely apt to be present where blood, or the products of exudation or secretion, are undergoing absorption;—does it not appear probable, moreover, from the lucid description given by Mr. Paget (when he says, that during the formation of these corpuscles “they present a gradual increase of shining black-edged particles, like minute oil-drops, which accumulate in the cell-cavity, and increase in number, and sometimes in size also, till they fill it”) that these compound granular cells, when associated with inflammatory products, fulfil a very important function, as the media through which the liquefied, softened, and disintegrated products of inflammation are gradually absorbed?

The observations of Reinhardt, Dr. Andrew Clark, Paget, and Gairdner also place it beyond a doubt, that compound granular cells may result from a fatty degeneration of the textural cells of a part; just as calcareous or pigmental degenerations occur, and which are common to primordial cells. While there can be no doubt, therefore, that fatty degeneration of lymph or textural elements may lead to the appearance of compound granular cells, that process can scarcely be called degeneration which is associated with development, growth, and complete absorption, by which the indurated and confused parts of an inflammation, such as the solidified portions of a lung in pneumonia, are ultimately cleared up.

Degenerate products are usually persistent, but the compound granule-cell is not. It seems to have an important function to perform in the removal of fluid, effete, or softened exudations, after which it too disappears.

The most frequent and important result of inflammation is the formation of pus by the growth of pus-cells. If a phlegmon or boil be observed, when it is a firm, hard, and solid mass of texture and exudation, we may feel in a few days that the solid mass has become fluid, and that it has not increased in bulk. The solidity and hardness are due to the inflammatory changes and effusion, the softening is due to the growth of pus-cells developed from the germinal elements of surrounding tissue (VIRCHOW, BEALE). So it is with the cells of vesicular eruptions which become pustular. The new cells there also become pus-cells—a change which may be accomplished in twelve hours, or sooner (PAGET). The following circumstances point to the development of pus from pre-existing germinal matter, namely, that,—(1.) A preliminary lymph-cell cannot always be discerned; (2.) The modification of the suppurative process, which occurs in the inflammation of mucous surfaces, where the formation of pus seems at once to take the place of the natural cell-growth, without any apparent distinction or alteration of the membranes of the mucous cells, corresponding in this instance to the most simple idea one can have of what Virchow terms *parenchymatous inflammation*, as described at page 93. Ultimately the natural mucous secretion undergoes a change. The characteristic cells on its surface drop off in all stages of abortion. Impaired co-





pus-cells, but multiform mixtures of withered cells appear, with molecular and fatty matter, escaped and shrivelled nuclei, blood-corpuscles, and fragments of granular matter like shreds of fibrine. The *liquor puris* becomes unduly liquid, and the pus is then said to be *watery* or *ichorous*. It may even, in weak and tuberculous patients, consist chiefly of a thin serum, mixed with flakes or curdled, when it has been called *serous pus*. When the coloring matter of blood is mixed with it, it is called *sanious pus*. Chemical or vital changes of various kinds bring about a peculiar decomposition in pus while yet in contact with living parts, although it is probable that atmospheric air, or gases from an internal cavity, may have to do with the change; but hydrosulphate of ammonia is frequently developed, especially in abscesses about the alimentary canal, near the tonsils or the rectum. The stench is then most offensive when the fluid is set free. Pus, besides possessing certain chemical properties, may possess certain specific properties: thus it may be impregnated with certain poisons, as that of syphilis, or of small-pox; it is also often, in certain constitutional states, loaded with foreign matters, such as urate of soda.

The formation of pus is termed *suppuration*. It takes place under three conditions; namely,—(1.) Circumscribed; (2.) Diffused; and (3.) Superficial.

As examples of the circumscribed formation of pus may be mentioned an *abscess*, a *boil*, or *phlegmon*, in which the suppuration is inclosed within a cavity whose walls are composed of connective areolar tissue, and into which interstitial exudation of inflammatory lymph and serum has extended over a certain area. It happens that while the central portion of an area has become purulent (*i. e.*, has produced pus-cells as a result of the continuous premature proliferation of tissue), the peripheral part has maintained its firmness and solidity by activity of nuclear growth—and sometimes a “thin, opaque, yellowish-white layer, easily detached,” separates the suppuration area from the denser part. This has been called a “*pyogenic membrane*,” from the supposition that its function is to secrete the pus, whereas the nuclei and cells of the denser part are growing by continuous but premature development into pus-cells. Abscesses are sometimes formed without any of the usual accompanying signs of inflammation being present. They are generally slowly formed, and are named *old* or *chronic* abscesses. When suppuration happens in the natural cavities of the body it is still circumscribed. It is not then, however, called an *abscess*, but a *purulent effusion*.

*Diffuse suppuration* is exemplified in *phlegmonous erysipelas*, or the *purulent infiltration* of an organ. In such cases the inflammation extends through a wide extent of tissue, and from first to last the boundaries are ill-defined. The growth of pus-cells is distinctly interstitial. They are generally rapidly formed, and the tissue becomes thoroughly infiltrated, as if soaked in pus. The usual want of cohesion in the elements of tissue involved in inflammation prevails from the first, and ultimately large *sloughs*, or death of portions of texture, may take place. In some textures of a loose kind it is believed that the pus may spread about or infiltrate



in their turn again provide themselves with dividing nuclei, and so the process of multiplication goes on.

Dr. D. R. Haldane, of Edinburgh, has observed and recorded the continuous development of pus-cells from the cylindrical variety of epithelium. In a case of small-pox, he found the larynx and trachea coated over with a soft, dirty-looking deposit, which was found to consist of pus-cells. On gently scraping the surface, the cells were found enlarged, and, in place of containing a single nucleus, each contained several—three, four, or more. These were derived from the proliferation of the original nucleus. External to the cells were young ones in all stages of development (*Edinburgh Medical Journal*, Nov., 1862, p. 439).

The more completely the epithelium is of the stratified kind, the less is the surface liable to ulceration (*e. g.*, the urethra in gonorrhoea); but those mucous surfaces where the epithelium is of the cylindrical form scarcely ever produce pus without ulceration (*e. g.*, the intestines). Pus-cells, mucous cells, and epithelial cells, are now regarded pathologically as equivalent elements, and which may replace one another; but physiologically they are not equivalent elements, inasmuch as they cannot perform each other's functions. Deeply seated pus-formation may proceed from *connective tissue*, or from the nuclei of vessels or sheaths of tissue. An enlargement of the *connective tissue* germs occurs (OTTO WEBER), which divide and subdivide, and so multiply excessively, by divisions of the larger germinal masses or cells. Round about the irritated or inflamed parts, where single cells lay, masses or groups of cells are formed, a large new formation grows, and towards the interior of this growth, heaps of little cells accumulate. These little accumulations occur at first as diffuse “infiltrations” of roundish masses, encircled by an intermediate growth, which continually liquefies as proliferation of the cells extend. Virchow regards this liquefaction as of a chemical nature; the intermediate substance (which yields gelatine) becomes transformed into mucus, and being ultimately converted into an albuminous fluid, is thus rendered liquid. Thus two different modes of pus-formation are distinguished, according as (1) the growth of the pus-cells proceeds from the germs of *superficial* tissue, like *epithelium*, or (2) from *connective tissue*; and two forms of inflammation can in like manner be separated from each other, namely—(1.) The *parenchymatous inflammation*, where the process runs its course in the interior of the tissue-elements (*e. g.*, connective tissue cells or germ masses, hepatic cells), without our being able to detect the presence of any free fluid which has escaped from the blood, but where softening and fluidity is due to the process above described. (2.) The *secretory* (exudative) *inflammation* of superficial tissue-elements, where an increased escape of fluid takes place from the blood, and conveys the new products of growth and altered secretion along with it to the surface.

The *parenchymatous inflammation* has from its outset a tendency to alter the elements of tissue and their special functions. Whereas the *secretory inflammation*, with a free exudation, in general affords a certain degree of relief to the part. Witness the relief which



neously in order to effect ulceration: (1.) An exudation of *inflammatory lymph* and *serum* surrounds the mass of young cells which constantly continue to grow and to break up (proliferation). (2.) Cells are thus continually growing on the surface, to be carried off by a fresh exudation. (3.) Liquefaction of the gelatinous interstitial material supervenes, and so destruction of tissue takes place continuously. Thus an ulcer forms.

**Granulation** is one of the modes in which a wound, or sore, or a part previously actually inflamed, heals. It is then said to do so by "second intention," and is always a reparative process. Granulation may occur with or without suppuration. The first mode is extremely common. The latter is occasionally seen in the healing of syphilitic maculæ and ulcers of the cornea, and Mr. Hunter conceives he once met with it in the union of a broken thigh bone.

Granulation is associated with an exudation of inflammatory lymph, into which old vessels extend, and new ones are formed. A new surface thus results, which is "granular"—the granule being a small conical tumor or growth, composed of a mesh of terminal loops, formed by capillary vessels shooting into the effused lymph. The figure and color of the granulation are determined by the state of the circulation; when that is feeble and inclined to stagnate, the granulation is broad, flat, and spongy, and either pale or of a livid hue; when, on the contrary, it is vigorous, the granulation is conical or acuminate, and of a bright-red tint (TRAVERS). The vessels prolonged into the granulation are more or less tortuous, and so numerous as to require a high magnifying power to exhibit their distinctness after successful injection. These vessels become contracted to obliteration as the period of cicatrization approaches. Granulation may take place from a surface, or from the sides of an abscess. If from the cutaneous tissue, the sore heals by a process of skinning; the skin always springing from the edges of the wound. Again, if granulations spring from the walls of an abscess, their opposite surfaces may unite. Granulations sometimes form with great rapidity. Mr. Hunter has seen, after trephining a patient, the dura mater strongly united to the scalp in twenty-four hours. Granulations, however, have not in all cases an equal disposition to unite. Thus the granulations of fistulous abscesses are little prone to adhere, their surfaces being often as difficult to unite as those of a mucous membrane; indeed, it is often impossible to produce adhesion except by exciting a considerable inflammation. A part having healed by granulation, uniformly contracts. This contractile force is so great that although the sore made by the amputation of a thigh is seldom less than seven or eight inches in diameter, yet the cicatrix left on healing is hardly more than an inch or an inch and a half. From this cause we find, in parts that have been the seat of abscess, a marked depression at the point of cicatrization.

The reproductive energy of parts which heal by granulation, however, is not great. It is rare that the original tissue is perfectly reproduced. No fat, for instance, is re-generated in ulcerated adipose tissues; a muscle being divided, unites by a cicatrix of connective tissue, no muscular fibre being reproduced; and a divided



Air, generated by a process of commencing putrefaction, is not unfrequently contained in the *phlyctenæ*, and gives, to the finger touching the part, a sensation of crepitation.

*Dry* mortification is a rare disease, and has sometimes been caused by the ergot of rye, or other diseased grain, used as food, giving rise to the disease known as *ergotism*. In the year 1716, dry mortification appears to have been, to a certain extent, epidemic at Orleans, fifty cases having been treated at the Hôtel Dieu of that city. Dodard described it as beginning generally in one or both feet, with pain, redness, and a sensation of heat or burning, like that produced by fire. At the end of some days, the part became cold, as black as charcoal, and as dry as if it had been passed through fire. Sometimes a line of separation was formed between the dead and the living parts, and the complete separation of the limb was effected by nature alone, and in one case, the thigh separated in this manner from the body, at the hip joint. In other cases amputation was necessary. Mr. Solly has given an interesting case of this description, which occurred in the practice of Mr. Bayley, of Odiham. The patient was a child, three years and seven months old, from whom, by this spontaneous process of nature, both arms were removed above the elbow, the left leg below the middle of the thigh, and the right foot above the ankle joint, being a remarkable instance, in modern times, of this destructive disease (see "*Ergotism*," and *Med.-Chir. Trans.*, vol. xxii, p. 23).

The bones, the brain, the lungs, the liver, the spleen, and the kidney are all liable to *sphacelus* and *gangrene*; so are the different tissues, as the areolar, cutaneous, nervous, and serous. The muscles, tendons, aponeuroses, and bloodvessels are likewise all liable, but in a less degree.

### *Local and General Symptoms of Inflammation.*

Redness, or at least increased afflux of blood, swelling, or at least increased textural productivity, pain, throbbing, increased sensibility, disorder of function, arrest and change of secretion, are the phenomena which are associated with the local morbid state, or with the textures in its immediate vicinity. Increased local heat under all circumstances is constant. This has been recently proved to demonstration by the ingenious experiments of Mr. Simon, and his colleague, Dr. Edmund Montgomery (*A System of Surgery*, edited by T. Holmes, M.A., vol. i, p. 42). If the local process of inflammation, however, is carried on upon a minute scale, or in certain tissues, one or other or more of these symptoms may be absent; if, on the other hand, the local process proceeds on an extensive scale, and involves important and delicate textures of vital importance, then we have much more unequivocal expression given, not only to local symptoms, but to complex morbid processes affecting the constitution generally. Of these the chief are:

**I. Inflammatory Fever.**—Of the *constitutional symptoms*, as they are termed, the most prominent are those which indicate "*inflammatory fever, symptomatic fever, sympathetic fever.*" These constitu-





or the edges and central tip may be red and dry: the latter is probably the more frequent combination. (5.) *The Secerning*. The secretions and excretions in general are materially diminished. The bowels are constipated—mainly from want of mucous secretion from their lining membrane; the skin is hot and dry; the mouth is parched; the urine is scanty, high-colored, generally acid, sparingly aqueous, and holding much saline matter, with comparatively little urea, in solution. (6.) *The Nutritive*. Digestion is interrupted; so is assimilation; as the fever advances, so does emaciation; and strength is more and more prostrate.

The chilliness, often amounting to shivering, marks the *date* of the febrile disturbance; and rigors more frequently attend the commencement of spontaneous inflammation than of inflammation caused by external injury.

Regarding the constitutional state characteristic of inflammatory fever, some important general conclusions, especially insisted on by Dr. Alison and Dr. Watson, may be thus shortly stated:

(1.) It is to be observed that there is no fixed relation between the degree or intensity of internal inflammations and the constitutional fever attending them; nor is the fever always proportioned in its degree of violence to either the size or importance of the part inflamed. In some cases, writes Dr. Alison, where we are sure that we have had inflammation going on under our inspection, to extensive effusion of pus, the pulse has been feeble, the skin cool and damp, and the patient exhausted and faint on the slightest exertion; while in others there is high and more inflammatory fever, and in some of these the organ inflamed has been so to no extent, and its function comparatively little affected, but yet the patient has become comatose nearly as in typhus, and died so. Laennec makes an observation of a similar kind (*Edin. Med. Journal*, May, 1857); and Dr. Watson observes that the fever may be high and very strongly marked in that common complaint, the *quinsy*, *cynanche tonsillaris*, or *tonsillia*, which can scarcely ever be said to imply much danger. (2.) The situation, the extent, and the degree of the local inflammation being the same, the fever commonly runs higher in young and in plethoric persons, and in those of sanguine temperament, than under opposite conditions. (3.) Inflammatory fever is modified in its expression, and especially in the characters of the pulse, by the nature of the part which is inflamed. This has been already alluded to in regard to inflammations of the abdomen, where the action of the heart is depressed, and the pulse is changed accordingly, tending to death by asthenia; and also in regard to the brain, when the mode of death tends to be by coma, the pulse being slow, labored, and full. (4.) The type of the inflammatory fever is very much modified by constitutional circumstances, such as the previous habits of the patient, and whether any zymotic disease is associated with the local inflammation. (5.) The inflammatory fever undergoes a further change of type (*a*) when suppuration takes place; (*b*) when it continues long; and (*c*) when mortification or gangrene occurs to a large extent. (6.) The febrile state follows generally the local disease; but (7) there is also good reason to believe that the *pyrexial*



or *ataxic*. The tongue becomes dry, black, and tremulous, sordes cover the teeth and harden on the lips and angles of the mouth. Low muttering delirium, stupor, or coma prevail; tremors affect the voluntary muscles, and the fæces and urine pass unnoticed. This form of fever sets in as a consequence of some untoward or unhealthy tendency of the inflammatory process, such as when mortification of the part occurs. Any cause, however, by which the system becomes extensively vitiated will bring about this form of fever. It is not necessary that the part should die. Putrescence of the infiltrated exudations in the inflamed part, degenerating and decomposing, poison the fluids circulating amongst them, and so, by absorption, may induce the typhoid state. If this happens with an internal organ, the event is generally indicated by a sudden cessation of all pain, at which the patient often appears very happy, and even joyous, while to the experienced physician its sudden cessation is assuredly an evil omen (Watson). The most important vital functions are deeply impaired by a prolonged existence of this type of fever. It tends to death by a complete sinking of the circulation, and diminution and loss of animal heat; or deepening stupor, with oppressed respiration, supervenes; or the patient dies by a combination of both conditions,—*asthenia* and *coma*.

III. *Hectic Fever*.—If suppuration continues beyond the powers of the constitution to supply the process with material to form inflammatory lymph and pus—if the inflammation continues, and becomes chronic as to time, inflammatory lymph continuing to be exuded, and pus continuing to form in profuse quantity, especially if an internal organ is its site—another type of *febrile* symptoms is apt to supervene, constituting *hectic fever*. It is not to be supposed, however, as was once believed and taught, that *hectic fever* is due, in every case in which it occurs, to the continued formation of pus. There are forms of *hectic fever* unconnected with suppuration anywhere, but associated with some analogous wasting of the bodily substance; for example, a prolonged secretion of milk in mothers who suckle their infants beyond the natural period. In all cases where a drain upon the system is established beyond its means, such a complex morbid condition of the body as *hectic fever* may be thus induced, and the mischief may not be revealed by any other symptoms. This type is particularly distinguished from the inflammatory and typhoid forms of fever by its remarkable intermissions, which are usually periodical; a period of remission and a period of exacerbation usually occurring once, and sometimes twice, in the twenty-four hours. It is also characterized by an excessive waste of the tissues of the body; and the sweating which attends the paroxysms causes great exhaustion. The assimilative and nervous functions are comparatively unimpaired, so that it is a febrile state generally of very long continuance. The mind remains perfectly clear—often vigorous and active—even when the body is debilitated; and if the intervals between the paroxysms are tolerably free from febrile excitement, the *hectic* type of fever may be protracted much beyond what at first sight might appear credible; and thus it is sometimes within our power to alleviate



emaciated. It is then that *diarrhœa* is apt to supervene, and to aggravate the sweating, so as completely to exhaust the remaining strength. The mind, unclouded before, gently wanders now, and the functions of life cease, generally without a struggle. It is often one of the closing symptoms, most strongly marked, in pulmonary consumption; and the non-professional pen of our great novelist, Mr. Charles Dickens, has beautifully portrayed its more striking features in the death of Smike:

“But there were times, and often too, when the sunken eye was too bright, the hollow cheek too flushed, the breath too thick and heavy in its course, the frame too feeble and exhausted, to escape their regard and notice. There is a dread disease which so prepares its victims, as it were, for death; which so refines it of its grosser aspect, and throws around familiar looks unearthly indications of the coming change,—a dread disease, in which the struggle between soul and body is so gradual, quiet, and solemn, and the result so sure, that day by day and grain by grain the mortal part wastes and withers away, so that the spirit grows light and sanguine with its lightening load; and feeling immortality at hand, deems it but a new term of mortal life,—a disease in which death and life are so strangely blended that death takes the glow and hue of life, and life the gaunt and grisly form of death.”

The forms of fever now noticed, as phenomena which may be associated with the inflammatory process, are to be regarded as various types which the febrile state may assume.

### SECTION III.—DEGENERATION OF TISSUE.

**Definition.**—*Degeneration of tissue implies such a departure from the normal state as would give rise to a palpable appearance in its minute elements of a granular disintegration or detritus; or of any deterioration which, by the functional actions of repair in the normal state, could not have been left there, nor been visible.*

**Pathology.**—The circumstances under which degenerations occur are of the nature of decay and death. For example, degeneration occurs to an immense extent in the tissues of the aged, especially in the heart and arteries, and to a less extent in the voluntary muscles and the hard textures. Towards the close of the life of a part of the body, degeneration takes place—as, for example, in the textures of the placenta when utero-gestation is nearly complete. To such degenerations Virchow has given the name of *necrobiosis*, because death and degeneration seem to be brought about by altered life at the close of natural existence. In this respect it may be truly said, that “As we begin to live we begin to die.” A spontaneous wearing out of living parts goes on, so that destruction and annihilation are immediately consequent upon life. *Softening* is the ultimate result of such degeneration, which becomes palpable chiefly by the decided friability of the parts. The minute elements of tissue lose their coherence, and at last really liquefy, so that pulpy or fluid products take their place. When it is remembered, also, how abundantly a gran-



In every texture the degeneration becomes evident in a similar manner. Isolated, extremely minute globules of fat appear in the cell-contents, and, becoming more abundant, they gradually fill up the cell-cavity. Usually the fat-granules appear at some distance from the nucleus; but ultimately they lie as close to each other as in the *colostrum corpuscles* of milk. At last the nucleus is no longer visible, and the membrane of the cell finally disappears—probably by a species of solution. If the degeneration occurs in the more rigid structures, as, for example, in the walls of arteries, the fatty granules retain the form of the cell which they replace. Such degeneration in arteries is first seen in the connective tissue corpuscles composing the innermost layer of the internal coat. Afterwards the intermediate substance softens, the degenerate fat-granule masses fall asunder, and the current of blood may carry away the particles of fat with it. Thus a number of uneven places (cicatricial-like loss of tissue) may be produced upon the surface of the larger vessels, without the appearance of ulceration (VIRCHOW).

In fatty degeneration of the substance of the heart, there is discoloration of its whole substance. It assumes generally a pale, yellow hue, with peculiar spots on the papillary muscles. Short, yellow streaks, which communicate with each other, are to be seen in the direction of the primitive fasciculi, and pervading the substance of the papillary muscles.

Yellow softening of the brain is a form of fatty degeneration, and this yellowness is due to the accumulation of finely granular fat. At every point where fatty degeneration attains a high pitch, great opacity will always present itself. A transparent part becomes opaque as in the cornea, where the fatty clouding marks the *arcus senilis*, described by the late Mr. Canton, in persons past middle life, and which has been regarded as an index to the existence of fatty degeneration of other more important organs, although the importance of the sign may have been exaggerated. In some form of Bright's disease, the uriniferous tubules become filled with fattily degenerated epithelium, which appears on the surface as opaque spots.

Additional examples of this fatty degeneration, are to be seen in the *fatty liver*, and in *mollities ossium*, atrophied renal capsules, and thymus gland, and the muscles—voluntary as well as involuntary—the fatty degenerations of the placenta, of cartilage, of bone, and of morbid growth: indeed, there is no kind of tissue, healthy or morbid, which may not undergo fatty degeneration.

When the normal structure of the part is thus transformed into fat, it is ultimately destroyed, and the place of the histological elements is gradually occupied by a purely emulsive mass—a kind of milk or fatty *débris*—that is, an amorphous accumulation of fatty particles in a more or less highly albuminous fluid (VIRCHOW).

With reference to fatty *degeneration* in particular organs, see the account given of local diseases.





The degeneration may follow upon the metastasis of calcareous salts, not excreted by the kidneys, in cases of caries of the bones, necrosis, or osseous cancer. I have seen specimens in the most interesting collection of Professor Virchow which show that metastatic deposits of bone-earth have taken place in the lungs and in the stomach under such circumstances. Considerable portions of the pulmonary tissue were *calcified* or *petrified*, without any apparent injury to the permeability of the respiratory passages. The lesion in the lung looked like a portion of fine bathing sponge. The mucous membrane of the stomach was in like manner transformed into a *calcified* or *petrified* mass. It felt like a rasp, and grated under the knife, so that the stomach-tubes seemed imbedded in a stiffened mass. The basis of such degeneration, in which the lime-salts find a resting-place, are the fine fibrous or connective tissues; and hence the degeneration is seen to occur in fibrous tumors, in serous membranes, in the parenchyma of lungs and stomach (as in the instance just mentioned), in cicatrix tissue on the skin, in the valves of the heart, in the connective tissue of muscle sheath, as well of the heart as of common muscle; in the tunica albuginea, in the fibrine coagula, in the heart's cavities, in aneurismal sacs, and in the thyroid and pineal glands. The cretifications of fibrine, of pus, of tubercle, of cancer, of vegetations, of coagula, all pertain to this form of degeneration; and the process may be traced through all stages of progressive degeneration from the pulp-like condition to cement-like, compact, calculous concretion, as in the phlebolite of veins; also in the turbid, chalky, speedily condensing juice of the cysts of the choroid plexus, and the cell-incrustations of the pineal gland concretions, as well as in the calcification of *sarcomata* and cancers. With regard to the degeneration as seen in tumors, Mr. Paget describes two methods by which it advances, namely, a *peripheral* and an *interstitial* calcification. The former is the rarer of the two. In this form of degeneration the fibrous tumor is seen to be coated with a thin, rough, nodulated layer of chalky or bone-like substance. In the interstitial form the degeneration is interspersed throughout the tumor, and so arranged that by maceration a heavy hard mass is obtained, variously knotted and branched, like a lump of hard coral (Paget, *Surgical Pathology*, vol. ii, p. 139.)

(c.) *Pigment-Degeneration—Pigmentation.*

In this degeneration *pigment* takes the place of the minute tissue-elements, as fat or lime did in the previously described conditions. It is seen in mucus-corpuscles, as in catarrhal pneumonia, in the pulmonary epithelium, in the acini of the liver, in the epidermic tissue, in the corpuscles of the blood in ague and *melanæmia*. As in the former degeneration, so in this one, a distinction must be carefully made between fat-granule cells and pigmentation, for in both cases apparently the same image is offered to view.

The fat-granule cells appear as brownish-yellow corpuscles, but their individual particles have no positive color; whereas the pigment-cells contain unquestionable gray, brown, or black molecules



The contamination of the blood in these cases seems due to a degeneration commencing in the spleen.

In post-mortem lesions, the textures are thus seen to be very variously tinted, red, yellow, brown, green, or black, generally resulting from chemical alteration in the coloring matter of the blood or bile. The red pigments, as a rule, are due to the altered hæmatine, originally of a yellow color; and which is the common origin of three different kinds of crystals,—(1.) Crystals of *Hæmatoidine* are the most frequent products of blood-degeneration (VIRCHOW). These are formed spontaneously in the body, out of hæmatine; and in their most perfect form present the shape of oblique rhombic columns, of a yellow-red color, or, in thicker pieces, of a deep ruby-red. In little plates it frequently bears a considerable resemblance to uric acid. In the majority of cases, the crystals are of extreme minuteness—difficult to resolve, even with a power of 300 diameters. They are insoluble in alcohol, ether, dilute mineral acids, and alkalies; and exhibit a peculiar play of green, blue, rose-tint, and yellow colors, under the action of concentrated mineral acids. If large masses of extravasated blood continue to lie for any length of time, this is the substance into which the blood is transformed. An apoplectic clot in the brain, for example, is repaired by a large portion of the blood undergoing this transformation, and the color of the resulting cicatrix is due to the crystals of *hæmatoidine*. When a young woman menstruates, also, the cavity of the Graëfian vesicle, from which the ovum escaped, becomes filled with coagulated blood, and ultimately *hæmatoidine* crystals are the last memorials of the event (VIRCHOW). *Hæmatoidine* is also allied to the coloring matter of the bile.

(2.) Crystals of *Hæmine*, arising out of *hæmatine*, differ from *hæmatoidine* in this, that hitherto they are only known as artificial products, which have not yet been seen in the human body. They are of a dark-brown color. (3.) Rectangular crystals or spicules of *Hæmato-crystalline*.

The yellow pigments are due to blood very much dissolved or dispersed, as in ecchymosis, or to bile, when it is absorbed in the blood, and tinges all the textures. Coloring matter due to bile may be recognized in the urine, by the play of colors it gives with nitric acid. A small quantity of acid gives a green hue; and, as more acid is added, blue, purple, violet, and a red or brown yellow color will ultimately appear. Of the brown and dark pigments, there are two kinds. One kind loses color on the addition of nitromuriatic acid or chlorine water; the other resists not only these agents, but even the action of the blow-pipe. This latter pigment consists of carbon. The former is a peculiar secretion formed within cells, or is a transformation of the coloring matter of the blood (BENNETT). Blue and purple pigments have been seen in urine containing *uroxanthin*, or the *Indican* of Schunk; and illustrate the close connection subsisting between animal and vegetable coloring matters (Parkes *On Urine*, p. 198). For much more interesting observations on the nature of pigmentation, consult Bennett's *Principles and Practice of Medicine*, p. 249.



Such are the names, derived from appearances generally, under which the peculiar degeneration has been described, before microscopic examination demonstrated the structures implicated.

Chemistry and micro-chemical investigations have modified the views regarding the nature of the disease, and now and then have led to modifications in the nomenclature. Under this kind of inquisitive investigation it has been described, (1.) By Virchow under the name of "*animal amyloid*," he believing, from the behavior of the transformed substance with iodine and sulphuric acid, that the substance must be classified with the vegetable carbohydrates—cellulose and starch. (2.) Meckel retains the name of "*lardaceous*" or "*cholesterine disease*," believing that the essential character of the degeneration consists in the development of a peculiar fatty or lardaceous matter, of the nature of cholesterine. (3.) The more extended and definite examinations by Friedreich and Kekulé have shown that the substance of the purest amyloid degeneration more closely resembles the *albuminous principles* than any other substance we know of; and (4.) Schmidt has arrived at the same conclusion; [and also Pavy (*Guy's Hospital Reports*, 1864).] The question, therefore, is not yet definitely settled as to the exact nature of the substance into which the tissues are transformed in the so-called *amyloid degeneration*, but the weight of evidence points to its being *albumen* in some form; and the albuminoid deposits in the spleen of children, so well described by Dr. Jenner, must be classed as examples of this degeneration, and probably also the special lesions in *rickets*.

Investigations relating to amyloid degenerations have taken especially two directions. Pathologists have endeavored, (1.) To trace the extension of the process of degeneration throughout various tissues and organs of the body; (2.) To determine the essential nature of the material into which the tissue is converted.

It was Professor Virchow who first turned the inquiry into the direction it has now taken, and which has given a remarkable interest to the micro-chemical investigation of the substance into which the minute elements of the tissues and organs are transformed in amyloid degeneration. Virchow stated that the large Malpighian sacculi in the spleen (which, in some instances, looked like boiled grains of sago), were sometimes composed of a substance which gave the chemical reactions of cellulose, as seen in plants. Cellulose and starch are both vegetable constituents—"isomeric" forms of some common material; and what gave special interest to the observation of Virchow was the discovery that cellulose is also an element in the covering or skin of the "*Tunicata*"—a genus of *acephalous mollusca*—and therefore not a constituent of only vegetable organization.

This discovery of cellulose in animal tissue induced Virchow to look for it or its analogue—namely, "starch"—in the human subject. He recognized it in the *corpora amylacea* of the brain. These contain a substance chemically related to starch or cellulose; and these bodies were first seen and named by Purkinje, who gave them the name they have, not on account of chemical characters,



“amyloid matter,” “zoo-amyline,” or “animal starch.” It owes its origin, not to any direct function of the organ, but its formation seems to take place almost immediately upon contact with albuminous matter, when this remarkable product is the result and which may be obtained as a white powder. It seems capable of being produced in greatest abundance by the hepatic tissue; but its formation may proceed at any part of the vascular capillary system. If, therefore, it is thus formed normally, it may also be formed, retained, or transformed in a morbid way. In *diabetes* we have an instance of the transformation of the product into sugar at the expense of the tissues at large; and which is so discharged by the urine.

The amyloid degeneration we are now considering has thus had various names to denote its presumed chemical nature, namely,—(1.) *Cellulose degeneration*; (2.) *Amyloid degeneration*; (3.) *Cholesterine disease*;\* and now (4.) *Albuminoid degeneration*.

The analysis of the pure matter is very defective. Such as it is, it shows the substance to be *albuminoid*, and combined with *nitrogen* rather than *starch*; and those who describe the reaction of *cellulose* and *starch* with iodine and sulphuric acid, seem only to agree with each other in giving singularly diversified descriptions of color, which, perhaps, to those familiar with the writings of the late Dr. George Wilson, on color-blindness, may be accounted for. Such diversity may be explained in some measure, also, by the fact that the degree of concentration of the reagents materially concerns the results; for, as Virchow correctly observes, the blue coloration is only got after a considerable period, and in practised hands, and it may pass from a bright purple to a very blue color. Nevertheless, the action of iodine solution on the so-called amyloid tissue is peculiar and definite, independently of a blue color.

The appearance of a chemical reaction, which gives a hue different from the mere dyeing with the iodine, and which *suddenly* deepens in tone, from the moment it begins to take effect, to a deep brown-red color, is sufficiently characteristic. When this takes place with the solution of iodine ALONE, it distinguishes at once the substance from *cellulose* and *cholesterine*. The following tabular statement will show the differences more clearly:

CHOLESTERINE.	AMYLOID OR ALBUMINOID.
1. Unchanged in color by iodine alone.	1. Immediate coloration (of the nature of a reaction) by iodine alone.
2. Insoluble in water.	2. Dissolves in warm or boiling water. (Boil sections in a test tube.)
3. Melts with heat.	3. Does not melt with heat—only dries up, and still gives the same reactions with iodine.
4. Passes into a brown fluid on the addition of sulphuric acid concentrated.	4. Swells in, but does not dissolve with sulphuric acid, with change of color.
5. Soluble in ether.	5. Not soluble in ether.

\* [Meckel first asserted that the deposit consisted essentially of cholesterine. This substance is not nitrogenous, and does not give the characteristic reaction with iodine, and has only been found in the liver in connection with the waxy deposit, and is, probably, an accidental associate, where fatty degeneration is coëxistent.]



By way of chemical analysis, very trustworthy results seem to have been arrived at by Friedreich and Kekulé. On submitting the white amyloid matter to ultimate analysis, they obtained the following composition in equivalents per cent. (*Med.-Chir. Review*, 1861, p. 59.)

	C.	H.	N.
<i>Amyloid</i> , . . . .	= 53.58 . . .	7.0 . . .	15.04

Now, the composition of albumen, according to Dumas and Cahours, Lieberkühn, and Rüling, is as follows:

	C.	H.	N.
<i>Albumen</i> , . . . .	= 53.5 . . .	7.1 . . .	15.8
Dumas and Cahours,	{ 53.4 . . .	7.2 . . .	15.7
	{ 53.5 . . .	7.8 . . .	15.7
Lieberkühn, . . .	53.5 . . .	7.0 . . .	15.6
Rüling, . . . .	53.8 . . .	7.1 . . .	15.5

Surely these results show an almost perfect chemical identity between albumen and the morbid substance found in the so-called waxy spleen; and demonstrate that the waxy degeneration, in the spleen at least, is due to a peculiarly modified albuminous material, and not to starch? On the other hand, the chemistry of the corpuscular variety of the *corpora amylacea* occurring as a deposit in various parts—*e. g.*, in the brain, the prostate, and the *ependyma* of the ventricles—shows a reaction almost identical with starch. The corpuscles also have concentric laminæ, and, according to some, resemble starch-granules when polarized. As regards the corpuscles of the prostate, sugar has been chemically produced from them, and demonstrated by Trommer's test.

Many of these corpuscular varieties of amylaceous bodies are no doubt of the same nature as starch; and therefore the direction which inquiry ought now to take, will be to determine "Whether or not there is any chemical affinity on the part of the formless matter of waxy degenerations with the corpuscular variety of the amylaceous concretions?" Such an affinity has been assumed hitherto; but, so far as observation has gone, the evidence of any affinity seems to be getting less and less. On the other hand, the modifying effects of admixture and of growth, are very remarkable as regards these prostatic concretions. Some of them *iodine* will not color blue, not even after sulphuric acid has been added; and as growth proceeds, any amyloid matter they contain gradually disappears. Many admixtures of organic and inorganic substances give various shades of color; and the yellow-brown colored deposits failed to give forth sugar to Paulizky's attempts.

[Dr. W. H. Dickinson, after very elaborate investigations of the so-called amyloid substance, has reached the following conclusions: That the deposit essentially consists of an outpouring of a certain material which differs from the proper constituents of the body; that this material has no affinities with starch, but is essentially fibrous, and has been deposited in this form in consequence of the loss of the alkali with which it is ordinarily combined, and which seems to be necessary to hold it in solution—

**Beasley's Druggists' General Receipt Book**  
 And Veterinary Formulary. The seventh American edition.  
 Price, \$3.50.

**Birch, on Constipated Bowels,**  
 The various causes of the disease, and modes of treatment.  
 Third edition, 12mo., cloth, \$1.25.

**Wythe's Physicians' Pocket Dose and Symptom Book.**  
 Eighth edition, 32mo., cloth. Price, \$1.00.  
 do. do. leather, with tucks and pockets, \$1.25.

**Cleaveland's Pronouncing Medical Lexicon,**  
 A new and improved edition (the eleventh).  
 Price, \$1.25.

**Richardson's Practical Treatise on Mechanical Dentistry.**  
 A new and much enlarged edition, containing 150 Illustrations.  
 Bound in Leather. Price, \$4.50.

**Taft's Practical Treatise on Operative Dentistry.**  
 A new and thoroughly revised edition, with over 100 Illustrations.  
 Price, bound in leather, \$4.50.

**Robertson's Manual on Extracting Teeth,**  
 With Illustrations. A new revised edition.  
 Price, \$1.50.

**Goff's Physicians' Combined Day Book, Ledger and Register**  
 For Patients. (For plan and description see Prospectus in general Catalogue.) A quarto volume, strongly bound.  
 Price, \$12.00.

## A FULL DESCRIPTIVE CATALOGUE

Of all our Publications, with prices attached, furnished free by Mail or otherwise, upon application.

LINDSAY & BLAKISTON, *Publishers,*  
 25 SOUTH SIXTH ST., PHILADELPHIA.



**“dealkalized fibrine.”** This is shown by the results of ultimate analysis of this morbid deposit, which make it agree in composition with fibrine and albumen. That it is fibrine and not albumen, is proved by its strong tendency to undergo contraction after its deposition. That it becomes converted into fibroid tissue, a metamorphosis which is common with fibrine whenever it is deposited in small bulk, as a coagulum in the arachnoid, or vegetations upon the valves of the heart. That in certain cases it is identical in appearance and reaction, as well as continuous in position, with the hyaline casts which are found in the renal tubes, the fibrinous nature of which it is not possible to doubt. A substance identical with it, and giving the characteristic color with iodine, can be made artificially out of fibrine by neutralizing or removing the alkali which the fibrine naturally contains. If potash or soda be added to the diseased tissue, thus artificially making a natural fibrine of it, it at once ceases to give the characteristic color with iodine. If a solution of sulphate of indigo be added to healthy tissue, the color of the solution is destroyed by virtue of the alkali contained in the tissues, but if the same solution be added to lardaceous degeneration, the color is vividly retained, because of the absence of alkali. An analysis of lardaceous liver shows a diminution by one-fourth of alkaline salts. While the alkalies are wanting in the morbid deposit, the earthy salts, as if to make up the deficiency, exist in larger quantity than in health (*Med. Chir. Trans.*, vol. i, 1867; *On the Pathology and Treatment of Albuminuria*, 1868).]

**General Characters and Anatomical Description of Tissues which have undergone Amyloid Degeneration.**—The cut surface of an organ so affected, has a semi-transparent appearance. It feels like a piece of soft wax, or of wax and lard combined (WILKS). It cuts into portions of the most regular shape, with sharp angles and smooth surfaces; and the thinnest possible slices may be removed by a sharp knife for microscopical examination, without any special preparation. The tissue is abnormally translucent. Water and alcohol, acids and alkalies do not produce any change upon the transformed parts, which may be kept for a length of time without decomposition. The organs affected are increased in volume, in solidity, and in weight, absolute and specific. Anæmia is predominant; but the color of blood or of tissue, shines through the semi-transparent morbid substance.

Amyloid degeneration is generally widely diffused; so much so, that a constitutional state of ill-health seems associated with its production; and in cases preceded by a local disease, such as caries of a bone, the degeneration may be found in the adjacent lymphatic glands only (BILLROTH). This is the earliest appearance of the degeneration yet recognized.

The small vessels of the tissue—the more minute arteries in particular—are, as a rule, the first structures attacked. The coats of the arteries become thickened and granular, and at last pellucid, transparent, and hyaline. Their calibre is reduced, and their cut section remains patulous.

It is the middle or muscular coat of the vessels which first changes. Each fibre-cell becomes a compact hyaline, pellucid, transparent particle, with an indistinct outline, and all the tissue involved becomes at last uniform, clear, and transparent. The degenerate



**Elements of Tissue in which Lardaceous degeneration has been demonstrated.**

1. Nervous System: ligamentum spinale cochleæ: atrophied parts of brain and spinal cord: gelatinous softening, and tumors.

2. Spleen: cells of the Malpighian sacculi and pulp: thickened walls of the arteries in all stages: the trabeculæ.

3. Liver: the hepatic cells and intralobular vessels (hepatic arteries): intercellular tissue.

4. Kidneys: Malpighian tufts and afferent vessels, the walls of which become enormously thickened: areolar tissue in the vicinity of the papillary ducts.

5. Muscular tissue of the heart and the uterus.

6. Bloodvessels of the villi and mucous membrane of the alimentary canal.

7. Osseous tissue.

8. Lymphatic glands.

9. Besides the original structures of the body, old deposits in serous membranes, having lost their fibrous character, becoming dense, more vascular and semi-transparent, undergo this metamorphosis (GAIRDNER).

10. Tubercle also becomes amyloid (GAIRDNER).

11. The cancerous nodules in a waxy liver also become amyloid (GAIRDNER).

12. In some cases of inflammation with exudation on the mucous membrane the exudation has assumed the amyloid degeneration (VIRCHOW).

13. The fibrine of a hæmatocele (FRIEDREICH).

The extensive range of organs in which this remarkable degeneration has now been demonstrated clearly shows that the lesion cannot be regarded as merely of local importance. Its occurrence seems rather to point to some general pathological state of which the degeneration is the expression. In the first instance it is found more particularly affecting the functional capillaries of the most important organs of the body—*e.g.*, the kidney, the liver, the spleen, the intestines, as well as the minute arteries of nutrition of those organs, and of the pia mater, bone, and lymphatic glands. The results of such a degeneration must therefore be sooner or later destructive,—(1.) To the function of the invaded organ; (2.) To its nutrition; and we can only arrive at a correct pathology of this degeneration by a close observation of the circumstances, condition, relations, and symptoms under which the lesion manifests itself. These must be studied especially in relation to the functional or physiological anatomy of the organs implicated. As yet the lesion has been recognized with certainty only in the dead-house. There it has been found associated with certain diseased states; and all the cases agree in this particular, namely, that the constitution of the patients, has

---

solution be poured upon a mucous surface or on the section of an organ, a uniform yellow tinge is procured; if this peculiar deposit is present, it becomes conspicuous by the contrast which its deep brown color presents to the unaffected parts (DICKINSON).]



deposited in the smallest arteries; the force of the circulation being lessened by distance, and broken by subdivision, the current has been sufficiently retarded to allow of coagulation or deposition. The deposit happening in the arteries and not in the veins, may be accounted for by the fact that venous blood is less rich in fibrine than arterial.

Albuminuria, especially when connected with nephritis, is the next most frequent circumstance of its occurrence. In 66 cases 4 were apparently due to this cause. A long continuance of albuminous urine must have an analogous effect to that of chronic suppuration. The albumen appears to carry alkali with it; albuminous urine, as a general rule, particularly in long-standing cases, is wanting in acid. In the 6 remaining cases there was no evidence of any morbid discharge, 3 of the subjects having been drunkards. Every process by which the blood is so altered as to contain excess of fibrine with deficiency of alkali is probably not yet known; other conditions unquestionably exist, which, impairing nutrition and altering the composition of the blood, may produce the effect of a chronic purulent discharge—the depurative deposit—without the medium of suppuration.

Dr. Dickinson's observations do not lead to the belief that syphilis has any direct power in the production of the deposit; nor is there any evidence that either cancer or tubercle are direct causes.]

Arguments are put forward by Virchow and Frerichs to show that the lesion may be due to a deposit from the blood; but the weight of evidence seems, on the whole, to point to a peculiar degeneration of existing tissue. (1.) In cases where the lesion follows affections of the bones, the lymphatic glands adjoining the diseased bones are implicated before the kidneys, liver, or mucous membrane of the intestines. (2.) General causes of ill-health (cachexia), pointing to impoverished blood, are in operation, and organs situated in different parts of the body are simultaneously affected. (3.) The fibrine of the blood itself has been observed to undergo the degeneration; for Friedreich found a substance which gave the amyloid reaction with iodine in the old fibrinous layer of the sac of a hæmatocele.

In this remarkable degeneration an acquaintance with a new fact in pathology must be recognized—*i. e.*, since 1854—associating itself with grave constitutional disease, and distinguished from every other morbid condition hitherto known, by the physical, chemical, and physiological characters just described.

**The Clinical History** of amyloid degeneration is remarkably deficient. The effect of the degeneration is to interfere with function of organs and nutrition of parts; and the injurious effects are the more marked as the lesion extends through many important organs. For example, hepatic cells cease to take part in the formation of sugar or the secretion of bile. Bloodvessels lose their power of transmitting fluid through their walls, and become impervious as to their canals. Hence those who suffer from amyloid disease have an appearance of *general ill-health*, denoted by paleness of the surface, by symptoms of anæmia, hydræmia, or by leukæmic affections of the blood; and the more so as the constitution is enfeebled by such morbid processes as ulceration of bones, syphilis, tuberculosis, or malaria. The sequence in which the different organs degenerate is uncertain. In most cases of caries and necrosis the kidneys seem





posed. It has been observed very frequently amongst the soldiers who have been dissected at the Military Hospital for invalids, formerly at Fort Pitt and now at Netley. The microscope and iodine test can alone determine its absence; and without microscopic examination the absence of the degeneration cannot be determined. For a detailed account of "amyloid degeneration" in the various organs, see the description given under LOCAL DISEASES.

Before stating the principles which dictate the treatment of the complex morbid processes just described, and of individual diseases in particular, it behooves the student, first, to make a separate study of *the varying types of diseases, their prevailing peculiarities, and the constitutional tendency to change of type which they assume at varying intervals of time*; and, second, to observe and learn to recognize *the various modes by which diseases terminate fatally*.

---

## CHAPTER IX.

### TYPES OF DISEASE AND THEIR TENDENCY TO CHANGE.

IN describing, appreciating, or ascertaining the type of a disease, our attention must be directed to a variety of phenomena and conditions; and the type of the disease only becomes characteristic and distinctive when some one or other of those conditions becomes predominant, or manifests itself more decidedly than others. The hereditary or natural constitution of the individual may be regarded as an important element in determining the type of the disease. Town life, as compared with country life, also exercises an influence on the type of many diseases; and there are good grounds for believing that the town life and artificial habits of the present period are more prejudicial to the strength of the constitution than those which prevailed when large towns were but rural villages, and the inhabitants more simple in their mode of life, and less artificial in their habits.

The occupation of the individual in many instances exercises an influence over the complex processes of disease; and there cannot be a doubt that some diseases have altogether disappeared, while others have been so much modified that their resemblance to the original form or type can with difficulty be recognized.

With regard to Edinburgh and its vicinity, Professor W. T. Gairdner observes that the *changes of type* which have occurred in epidemic fever, and especially in typhus fever, during the ten years previous to 1862, or since the cessation of the great epidemic of 1847-8, are not less remarkable than the diminution in the amount of this class of cases. The *relapsing fever*, or *synocha*, which formed so large a part of the epidemics of 1843-4 and 1847-8, has for the time absolutely disappeared. *Typhus fever* has become less fatal to



social, moral, and sanitary condition of the private soldier. Much expenditure has been incurred for the sake of enlarging and improving barracks, and in carrying out various recommendations of the House of Commons with respect to barracks and the hospitals connected with them. I am happy to say," continues the Right Hon. gentleman, "that these efforts have not been unattended with important results, as will appear from authentic returns of the mortality in the service. These returns have been prepared by the Director-General of the Army Medical Department, and I believe they are perfectly authentic, though it is certainly difficult to believe that so great a change can have taken place in so limited a period. It is possible that the greater youth of some portions of the army may, to a certain extent, affect the returns, but I believe the difference is mainly to be explained by improvements in the sanitary conditions under which they are now called on to serve.

"DEATHS AMONG THE TROOPS SERVING IN THE UNITED KINGDOM ANNUALLY PER 1000 OF MEN.

	From 1830 to 1836.	1859 to 1860.
Generally throughout, . . . . .	14	5
Cavalry of the Line, . . . . .	15	6
Royal Artillery, . . . . .	15	7
Foot Guards, . . . . .	21	9
	From 1836 to 1846.	1859 to 1860.
Infantry of the Line, . . . . .	17	8

"SIMILAR RETURNS FOR THE COLONIES ARE AS FOLLOWS:

	From 1837 to 1855.	1859 to 1861.
Gibraltar, . . . . .	22	9
Malta, . . . . .	18	14
Ionian Islands, . . . . .	27	9
Bermuda, . . . . .	85	11
Canada, . . . . .	20	10
Jamaica, . . . . .	128	17
Ceylon, . . . . .	74	27

"I have other returns from other colonies," continues the Right Honorable gentleman, "and I believe that these returns are authentic; and certainly they show how very considerable a diminution has taken place in the mortality of the army" (*Times*, March 4, 1862).

The late Lord Herbert was the main agent in accomplishing this great work, which, as years pass on, will become better appreciated, more widely known, and more energetically followed up.

Professor W. T. Gairdner has happily observed, that in proportion as we are getting rid of the severer forms of epidemic disease (*e. g.*, fever, dysentery, scurvy, influenza, all more or less preventible), which had deteriorated the health of the population previously to 1848, we are also getting rid of the more severe and unmanageable types of acute inflammation. Inflammatory diseases, like fevers, he therefore justly considers to be subject to epidemic causes of increase and diminution, both as regards frequency and severity; and he believes that the acute inflammations are nearly as much under the influence of the sanitary reformer as the more obviously epidemic fevers; and, further, that some even of the chronic organic diseases have already yielded, and may be expected still further to yield, to the improved habits, the better clothing, the greater abundance of



have been considered a sufficient tax for the strength of a man of upwards of five-and-twenty. Many of the features, also, which characterize the pathology of our age have arisen out of the treatment of infancy and childhood; and much of the deterioration of the race at large may be shown to date its origin from childhood. Thus, after seventeen years' observation in children's disorders, Dr. Pollitzer writes that *anæmia* and *chlorosis* occur alone or associated with *rickets*, *hypertrophy* of the *lymphatic glands*, and of the *spleen* and *liver*, to an incredible extent even from the first month of life. In the Children's Hospital at Vienna, from seventy to eighty per cent. he found to be thus affected. Wherever the nutrition of the child had been imperfect, the constitutional diseases associated with poverty of the blood became widely diffused. The stomach and intestinal tract first suffer, constituting the prevailing morbid condition of childhood—materially influencing the mortality at an early age, and if the age of childhood is survived, affecting the future health of youth and manhood, and doubtless of subsequent generations (*Med.-Chir. Review*, Report on Medicine, p. 261, July 1857).

The types of disease are also evidently modified by complication with other diseases, now more widely spread; and the doctrine of the incompatibility of two or more contagious diseases concurring in the same subject, has been clearly proved to be erroneous. Dr. Murchison, in an admirable paper on this subject in the *British and Foreign Medico-Chirurgical Review*, for July, 1859, has clearly shown the coexistence of *variola* and *scarlatina*; also, of *variola* and *rubeola*. Dr. F. J. Brown, of Rochester, has recorded a case of *variola* concurring with *measles* and *purpura*. The coexistence of *variola* and *roseola*, or *erysipelas*, of *variola* and *pertussis*, of *variola* and *varicella*, of *variola* and *vaccinia*, of *vaccinia* and *scarlet fever*, of *vaccinia* and *rubeola*, of *vaccinia* and *pertussis*, of *vaccinia* and *varicella*, of *rubeola* and *pertussis*, of *variola*, *rubeola*, and *pertussis*, of *scarlatina* and *rubeola*—the *rötheln* of the Germans—of *scarlatina* and *enteric* or *intestinal fever*, of *typhus* and *enteric*, *intestinal* or *typhoid fever*, have all been more or less clearly shown. Virchow relates a case of typhoid fever, combined with striking symptoms of cholera, occurring at Würzburg. Typhus fever and the marsh fevers have been observed to occur together (PRINGLE). Bilious remittents have prevailed with small-pox in the West Indies, forming, as an old writer remarks, "the most infernal combination that ever affected the human frame." Yellow fever has been associated with putrid typhus; while specific yellow fever and marsh fever, with phenomena similar in many respects to specific yellow fever, undoubtedly occur together.

There are good grounds for believing that as we approach certain well-marked geographical regions of the earth, where characteristic types of disease prevail, the confines of these disease-realms are found to mingle their types of disease together, so that the diseases of one region merge into and participate in many of the characters peculiar to the other. Cholera has extended its ravages over the earth, and is now a disease whose germs are endemic in our land;



in the course of time, and from causes not yet known. They have in fact undergone very considerable change since the early part of the present century; and it is on this account that inflammations of the lungs in particular are treated with equal success at present, with a much smaller loss of blood than they used to demand" (ALISON).

Such changes in the types of disease were formerly observed and much insisted upon by Sydenham, especially in the progress and recurrence of continued fevers; and it is now a fact well recognized, that not only does the prevalent mode of fatal termination during epidemic diseases vary, but so also do the types, peculiarities, and morbid constitutional tendencies vary in these diseases. It is chiefly with regard to the *local, sporadic, or intrinsic diseases*, and especially inflammations, such as the *cephalic*, the *pulmonic*, or the *enteric*, that any doubt exists as to whether or not they vary in their type. Distinct statements as to this fact, however, have now been made by many accurate observers, whose experience is of the utmost value to science. Dr. Alison and Dr. Bennett both agree as to the fact, "that of late years, and apparently also in different parts of the world, *inflammation*, the most important of all forms of *local diseases*, seldom shows itself with such general symptoms as demand or would justify, in the opinion of the practitioners treating them, or indeed could bear, the large bleedings which were formerly regarded as the appropriate remedy, and which accordingly are now seldom practised." There are not only also fewer examples of violent inflammation of the lungs to be met with but the *usual* (highly inflammatory) type of fever attending such inflammation has materially changed, as occurring in the present day. This change which has taken place in the *type* of the usual phenomena characteristic of inflammatory fever, cannot be explained merely by the circumstance that a previously enfeebled or diseased state of the system has brought it about in the individual. The inflammations of internal parts, such as pneumonia, now *occur often without febrile reaction*, and neither demanding nor bearing full bleedings, as described by Cullen and other authors. It is consistent, moreover, with the extensive experience of Drs. Alison, Christison, Watson, and many other physicians of the greatest eminence and long experience, that the inflammations now seldom occur with such severe symptoms of inflammatory fever as have been described at page 111, and which were the rule in the time of Cullen and of Gregory. The constitutional symptoms now attending such inflammations partake more of the type of the so-called *typhoid* state, and independent of any epidemic influence or poison having acted on the body.

The constitutional symptoms for many years past accompanying pneumonia, for instance, in this country, have been of the following kind: An enfeebled circulation; softness of the pulse, and easiness of depression by depletion, or even by taking the erect posture; tremors and feebleness of voluntary muscular motion approaching to subsultus; indifference to surrounding objects; sickness and vomiting in some cases, with dryness of tongue and lips in others; complete anorexia or depraved appetite; in all, the symptoms gen-





and demand a different treatment. Moreover, he observes that it is consistent with the experience of the veterinarians that the change in the type of disease has been observed among the lower animals to the same extent as in man.

In 1782, when Dr. Hamilton published his memoirs on the "*contagious catarrh*," or *influenza*, he records distinctly his belief that "our constitutions are considerably changed within the last century in Great Britain. Diseases," he remarks, "in their nature phlogistic (*e. g.*, *measles*), have appeared within the thirty years previous to 1782 less inflammatory than they formerly were, and accompanied with a considerable degree of putridity;" and bloodletting in the "*contagious catarrh*," he states with emphasis, cannot be tolerated. There seems, therefore, to have been even then an increasing belief that the degeneracy of the human race, as a whole, is in some respects advancing; and there seems some visible evidence of this more or less traceable through the past four generations.

Morel especially directs attention to the apparent increase in Europe of mental alienation, and of those abnormal states of existence which have special relations with the occurrence and existence of physical and moral degeneracy in the world; and if a comparative increase in the number of the insane cannot yet be proven, there would seem to be a tendency to more frequent complications among them of those morbid states which diminish the probability of cure, such as *general paralysis*, *epilepsy*, and a marked depression of all the intellectual and physical forces, which depression is consistent with the *asthenic* phase of present existence. *Hysteria* and *hypochondriasis*, formerly almost the exclusive appanage of the rich, the indolent, and those of a wasted life, are known to attack in great proportions the working and the agricultural classes, among whom suicidal tendencies prevail.

Dr. Forbes Winslow writes, with regard to nervous diseases, that cases of disease of the brain and nervous system are now not only of more frequent occurrence, but that a certain unfavorable type of cerebral disorder develops itself in the present age at a much earlier age than formerly. Softening of the brain, for instance, often manifests itself at the early age of thirty and thirty-five. The brain in the present day is overworked, its psychical functions are unduly exercised, strained, and taxed in the great effort required in the severe struggle and battle of life to obtain intellectual supremacy, professional emolument, and status (*Journal of Psychological Medicine*, July, 1857).

Morel again shows how imbecility, congenital or early acquired idiocy, and other more or less complete arrests of development of the body, and of the intellectual faculties, indicate the existence of children who have acquired the elements of their degeneracy during intra-uterine life. It behooves, then, all civilized governments anxiously to inquire into and to consider such facts as show,—(1.) The continued increase of suicide; (2.) The continued increase of crimes against order and law, or against the person; (3.) The monstrous precocity of young criminals; (4.) The abnormal conformations of the skull, and tendency to early union of the cranial sutures,



rise surely and progressively to the *degeneration* of the individual, are mainly demonstrable by the introduction of the following states, namely: persistent loss of appetite, indigestion, nausea; occasional diarrhoea, progressive emaciation, and cachexia; the appearance of pustular eruptions; the occurrence of eructations associated with offensive breath; serious disturbance of the function of the stomach, liver, kidneys, and heart, and the production of organic lesions in these organs, and in the structures of the bloodvessels, followed by fatal *serous effusions, dropsy, hemorrhages, extravasations, or apoplexies*. Intercurrent with these morbid conditions, at variable periods, and otherwise contingently, "fits of drunkenness," with sexual incompetency, different forms of psychical aberration, *delirium tremens*, suicidal melancholy, and such-like morbid phenomena ensue. Finally, epileptiform seizures, general paralysis, drivelling or slaver-ing idiocy, may close the scene. Those who become thus degenerate by alcoholic poison are arranged by Morel into two classes, namely,—*First*, Individuals who arrive at length, by a series of well-marked nervous lesions—physical and intellectual—at general paralysis. *Second*, Individuals who, although profoundly affected as regards enervation, still remain stationary at a certain state, leading a miserable existence, characterized *physically* by a special condition of ill-health (cachexia and marasmus), and *morally* by manifestation of the worst tendencies, and of the lowest animal propensities. The serious degenerative effects thus detailed in their extreme forms, resulting from the poison of alcohol, ultimately influence the pro-creative functions; first, by diminishing the vital standard of the offspring; and second, by annihilating the generative power altogether. When such results are coupled with the moral and social aberrations which ensue from bad example, misery, and want, in families and masses of men, they become ample sources of the degeneracy and degradation of the population, not only throughout the existing but succeeding generation; and not only is the vice of alcoholic abuse hereditarily transmissible (MOREL,) but it also frequently leads to insanity in the offspring of the drunkard (WHITEHEAD, ADAMS); and in cases where the tendency to alcoholic excesses has an hereditary origin, the cure of the dipsomaniac is generally impossible. Morel gives the following example, in which a well-marked succession of morbid phenomena became developed in different descendants of a family throughout four generations. The great-grandfather of the family was a dipsomaniac; and so complete was the transmission of the disease, that the race became totally extinct, under the well-marked phenomena of alcoholic poisoning and degeneracy. The effects entailed were: in the first generation, alcoholic excesses, immorality, depravity, brutish disposition; in the second generation, hereditary drunkenness, attacks of mania, general paralysis; in the third generation, sobriety prevailed, but hypochondriasis, lypomania, persistent ideas of persecution, and homicidal tendencies were expressed; in the fourth generation, intelligence was but feeble, mania became developed at sixteen years of age, stupidity running on to idiocy, and to a condition involving extinction of the race. Sir James Clark also made the observation, more than twenty



terations as regards not only its *physical*, but also what has been termed its "*Medical Constitution*." Fevers are known to change their types; epidemics to assume new tendencies; and inflammations and local lesions to affect in no constant manner the constitution of individuals at the same period, or at different times and in different countries. This view of the subject may be summed up in the eloquent language of Dr. Watson, when he writes,—“I am firmly persuaded, by my own observation, and by the records of medicine, that there are waves of time through which the *sthenic* and the *asthenic* characters of disease prevail in succession, and that we are at present living in one of its adynamic phases” (*Edinburgh Monthly Journal*, June, 1857). It must be admitted, however, that much of what is written and here quoted regarding changes of type is based on the uncertain impressions of individual men, sometimes handed down by tradition, rather than based on the results of a deliberate deductive inquiry from statistics applied to the question by men agreed as to the meaning of terms. On the other hand, I think Dr. Markham, in his very able Gulstonian Lectures on “The Uses of Bloodletting in Disease,” puts the evidence regarding change of type in disease on much too narrow a basis—not to say an erroneous one—when he holds that it is merely an excuse put forward for a change of practice in inflammations, and that the only argument in favor of such a theory rests upon the assumed conclusion that venesection produces different effects now from what it formerly did.

A somewhat similar view of this important subject has been ably advocated by Dr. J. H. Bennett, of Edinburgh. He contends that inflammation is the same now as it has ever been—that the analogy sought to be established between it and the varying types of fevers is fallacious—that we cannot place reliance on the recorded experience of the past—and that our recent changes in therapeutics are solely due to an advanced knowledge of diagnosis and pathology (*Principles and Practice of Medicine*, p. 267).

The local phenomena of inflammation are undoubtedly constant; but the question of change of type has reference to the constitution of the individual, and to the constitutional modes by which the inflammatory state is expressed in a given number of persons. Much of the argument of Drs. Markham and Bennett appears to me to be beside the question; and there are certainly good grounds for believing that elements of constitutional degeneration exist in such abundance, especially in the communities of large towns, that Dr. Watson has, I think, happily expressed the consequence of such deterioration in the sentence quoted from his writings on the subject.



appear to lose, with little anxiety, the consciousness of light and of ourselves." At such a time, the vivid impressions of a life well-spent, must constitute that *euthanasia*—that happy death—to be desired by all.

The untoward lesions or derangements of vital organs, which occur during the progress of disease, terminate the life of man by various modes of dying. While it is ordained that eventually all must die, yet it is possible sometimes to avert, for a time, the tendency to death. To know by what agents this may be properly accomplished, it is necessary to know the modes in which death may approach in disease. Dr. Watson has happily observed that life rests upon a tripod, whose three vital supports are, the *heart*, the *brain*, and the *lungs*. Through the impaired functions of some one or more of these organs, the tendency to death is expressed. The mode of dying may begin at the *head*, the *heart*, or the *lungs* (BICHAT). But inasmuch as the functions of these organs are mutually dependent upon each other, so impairment of function in any one of them may ultimately lead to death, while the mode of dying is expressed chiefly through the functions of another. The mode of dying in disease is usually a complex one, for many parts thus mutually dependent on each other, are more or less immediately involved. Therefore it is of the greatest practical importance to observe how and when the different functions begin to languish, and how they may be best sustained in their exertions to maintain life.

When a person loses blood to such an extent that he faints, as from a wound, or by hemorrhage occurring in disease, and if the flow of blood is not arrested, the state of *faint* or *syncope* continues, is not recovered from, and the heart's action ceases; not because it is unable to contract, but because its natural stimulus, the blood, is withdrawn from it, or does not arrive in sufficient quantity to be of use. This is called death by *anæmia*. In such cases, if blood can be timeously supplied to the heart (as by the operation of transfusion from a healthy person into the patient who is losing blood), the suspended function of the heart may be restored, and a supply of blood, sufficient to maintain life for a time, may be thus obtained.

The symptoms of approaching death by this mode of dying are, paleness of the countenance and lips, cold sweats, dimness of vision, dilated pupils, vertigo, a slow, weak, irregular pulse, and speedy insensibility. If the hemorrhage has been sudden, in large quantity, as from the uterus in "flooding," there may be nausea, or even vomiting, restlessness, tossing of the limbs, irregular sighing breathing (*anxietas*), delirium, and one or two convulsions before death ensues.

But another mode of death may be more immediately connected with the heart itself, and be independent of the supply of blood. In other words, the stimulus from blood may be sufficient, but the contractile power of the organ may fail. Such a mode of death is by *asthenia*. Many poisons act in this way, and many diseases which are due to morbid poisons in the blood tend to prove fatal





respiration be timeously resorted to, and persevered in. This prolonged action of the heart circulates blood, which is dark, venous, and not arterialized, and accordingly the face, at first flushed, becomes turgid, and then assumes a livid and purple hue; the veins of the head and neck swell, and the eyeballs protrude from their sockets. At length the heart ceases to beat, and life is extinct (WATSON).

Death by coma occurs when there is a loss of consciousness first, with the appearance of profound sleep, from which the patient may be partially roused. The symptoms of approaching death by this mode of dying consist in a gradual blunting of sensibility to outward impressions, slowness of respiration, the inspiratory effort being often delayed, and then performed with a sudden noise and jerking inspiratory effort, technically known as *stertorous* breathing. All voluntary attention to the act of breathing is lost, but the influence of a reflex stimulus to its performance continues. At length this function fails also. The chest ceases to expand, the blood is no longer aerated, and thenceforward precisely the same changes occur as in death by apnœa.

Such are the several modes by which death tends to approach; and “*to obviate the tendency to death*” is a doctrine which was often and strenuously inculcated by Cullen. After him, no less earnestly has it been impressed on many by my respected teacher, the late Professor Alison, whose interesting Lectures on *fevers* and *inflammation* furnished numerous illustrations. To his *Outlines of Pathology and Practice of Medicine*, and to the first volume of Dr. Watson’s *Lectures on the Principles and Practice of Physic*, the student is referred who would seek further information—sources whence the preceding observations have been mainly compiled relative to the modes by which death may approach:

“ Many are the ways that lead  
To his grim cave, all dismal; yet to sense  
More terrible at the entrance than within.”

“ It is as natural to die as to be born; ”

and thus, in

“ Passing thro’ Nature to Eternity,  
The sense of death is most in apprehension.”

---

## CHAPTER XI.

### PRINCIPLES WHICH DICTATE THE TREATMENT OF THE COMPLEX MORBID PROCESSES.

#### I. *As regards Fevers or the Febrile State.*

To avert the tendency to death in the febrile state, it is necessary to observe how fevers naturally terminate favorably. Four modes are enumerated by Dr. Parkes, namely:



The system ought to be supplied with an abundance of alkaline salts, if the urinary excretions are not eliminated. *Chloride of sodium, the alkaline salts of soda and of potash*, tend to aid the formation of urea and its elimination. Purgatives generally, and especially *salines*—i. e., salts of the alkaline and earthy metals—tend to insure a proper excretion, probably by removing from the blood some of the abnormal products formed in fever, and great relief may follow their moderate use. Where urea is retained they promote its elimination, because it is known that urea sometimes passes off by the mucous membrane of the intestines. But in some fevers, as in typhus, purgatives must be very cautiously and sparingly given, and always in mild doses. So also elimination by the skin, to the extent of *diaphoresis*, is to be dreaded in typhus fever. (See “Treatment of Typhus Fever.”)

The most important indication, however, in the management of the febrile state is to find some substance which, being “restorative” in its action (HEADLAND), will so act upon the blood, and thus restore the exhausted energies of the nervous centres. Food, mild stimulants, and quinine are all more or less employed; and quinine especially may be employed with benefit. Infusion of coffee as a medicine has been given by Dr. Parkes, with the beneficial effect of relieving headache. Böcker and Lehmann have shown that the use of coffee, in health, delays the metamorphoses of tissue, and excites the nervous system. The special treatment of the febrile state depends on the diseases of which it forms a part, and by which it is more or less modified—forming a special topic for consideration in the part which treats of special diseases. But it is above all necessary to guard against the habit of trying always to be doing *something*. As a routine system, nothing can be laid down as a rule, either in the direction of depletion, or of evacuants, or of stimulation. The febrile state is in many diseases part of the essence of the morbid state, which cannot be cut short nor materially subdued by remedies. There is no specific remedy for the cure of any fever; and in the present state of our knowledge regarding specific febrile diseases, there can be no specific remedy for their cure.

Every disease where fever is present, and every case of specific febrile disease, must be studied, so that its management or treatment may be regulated on the merits of the individual case; and must be regulated by the state of each particular function as determined by clinical investigation daily.

## II. As regards Inflammation.

It is necessary clearly to understand and to bear in mind that, in the first instance, it is not the *lesion* which may attend the inflammatory process as a result, which is to be attended to; but it is the *diseased action tending to the lesion* which it is the object of the physician to overcome, to subdue, and turn aside; and that the occurrence of any lesion is, if possible, to be prevented. It is to the *strictly vital action—the excitement of tissue*—which tends to the organic lesion, that remedies must be applied, in order to avert the



patient whom his master and teacher may wish to relieve. He may see the apoplectic sufferer roused to consciousness while the blood yet flows from the vein; and he may observe, also, that the *stounding pains* of the head in cephalic inflammations are immediately relieved, that the impatience of light and sound, the frequent, sharp, intermittent pulse, with vomiting or nausea on assuming the erect posture, the tendency to squint,—in short, all the urgent symptoms of incipient encephalitis, at once, or one by one, disappear as the blood continues to flow. He may also notice in *thoracic* inflammation that the pain, the dyspnoea, the tightness of the chest, all disappear. Dr. Alison, as regards pleurisy, and Dr. Watson, as regards inflammation of the bowels, bear personal testimony to the good effects of bloodletting. They experienced its sanative influence in their own persons, and the practice undoubtedly saved their valuable lives from these respective diseases. Testimony from such personal experience has also been borne by the late Dr. Gregory, of Edinburgh, and before him by the celebrated Dr. Radcliffe; and so also is the testimony of many who, having experienced the benefit of the remedy once, imagine that, when again attacked with inflammation, they may be again relieved by its use. Of any one of these illustrious examples from personal experience it might be said, as Dr. Gregory said of Dr. Radcliffe, that “he was at least no fool; and we may depend upon it he would not have allowed a hundred ounces of blood to be taken from him in one day without good reason for it” (*Edin. Med. Journal*, March, 1857).

“Although much has been done,” writes Dr. Alison, “particularly by the French pathologists, to enable us to judge of the texture within the chest which is the subject of inflammation, and although this is a matter of real importance, because we know that the history of the changes to be expected from inflammation in the bronchiæ, substance of the lungs, and pleura, is materially different, and of course the diagnosis of these gives us great advantage in studying the progress of any individual case,—yet as to the specific questions of bloodletting or not, the quantity, or the repetition of the bloodletting, our predecessors were very nearly as well informed as we are. It is an important practical error,” he also continues, “to fix the attention, particularly of students of the profession, too much on those characters of disease which are drawn from changes of structure already affected, and to trust too exclusively to these as the diagnostics of different diseases; because, in many instances, these characters are not clearly perceptible until the latest and least remediable stage of diseases. The very object of the most important practice, moreover, in many cases, is to prevent the occurrence of the changes on which these lesions depend. After these lesions are once established, the cases are very often hopeless, or admit only of palliative treatment. In those diseases in which most can be done by art, our practice must always be guided in part by conjecture, because, if we wait for certainty, we very often wait until the time for successful practice is past; and therefore, although an accurate knowledge of the whole history of each disease is essential to its proper treatment, yet, in a practical view, the most important part of its history is the *assemblage and succession of symptoms*, by which its nature, at least, if not its precise seat, may often be known, before any decided lesion of structure has occurred. Accordingly, when



seeing that it is *the diseased action which tends towards the lesion*—namely, effusion or growth of new material—which the physician desires to control by this remedy.

5. When the symptoms of *inflammatory fever* are little complicated and seen early, in persons previously healthy, the more violent the symptoms are, the more intense and rapid the constitutional reaction, if it does not indicate exhaustion; and the more decided the change on the function of the part affected, the more confidently we may depend on the effect of full bloodletting in relieving them.

6. When the symptoms of inflammatory fever have been uncertain and insidious in the beginning, so that the early stage has passed over unchecked, or modified by previously existing constitutional disease, or complicated with organic local disease, or when they denote debility, exhaustion, or the so-called typhoid state, they generally prove improper cases for bloodletting, even when seen within the first few days.

7. Generally, it may be stated, that when the fever is high (above 104° Fahr.), when we may be sure that over a part of the inflamed organ there is congestion, stagnation of blood, distension of vessels, commencing extravasation, and change of the constitution of the blood—but these latter changes still partial and not far advanced—the power of bloodletting to control the disease has been clearly established.

8. The nature of the membrane or organ affected must always be considered in estimating the propriety of bleeding. If a serous membrane, for instance, be actually inflamed, the patient, for the most part, bears bleeding well, and is usually greatly relieved by it. With respect to organs, it is found that inflammation of the brain is less influenced by bleeding than inflammation of the liver, and inflammation of the liver than inflammation of the lungs. The gist of the evidence brought forward in Dr. Markham's very able and interesting Gulstonian Lectures establishes the special efficacy of venesection in those inflammations which are accompanied with obstructions of the cardiac and pulmonary functions, but do not prove it to be useless nor injurious in the cure of acute inflammations generally, if its remedial functions are properly used, and the advantage gained be duly followed up by appropriate remedies, such as are to be presently noticed. The symptoms which demand a full bloodletting in pneumonia are also those which indicate the greatest danger—namely, violent pyrexia, usually beginning suddenly, with full, strong, hard, and quick pulse—urgent *dyspnœa*, even *orthopnœa*—swelling and flushing of the face, frequency and violence of cough, with scanty or truly pneumonic expectoration, aggravating the pain which extends through the chest,—when such symptoms are seen within *three* days of their commencement, especially in those of robust and full habit in the prime of life, bloodletting is the remedy to be used,—everything else is *trifling*, and it is *not safe* to dispense with it. The nearer a case answers this description, the more sure we may be that the effect of bloodletting will be satisfactory, and its repetition, if the symptoms shall recur, will be well borne (ALISON).





11. The proportion of the serum to the clot, and also its occasionally altered characters, are arguments for or against bleeding. When the quantity of serum is unusually large, unless the clot be very firm, bleeding ought not to be repeated. Also, when the properties of the serum are so altered that it coagulates and forms one mass with the clot, bleeding is constantly prejudicial; and, lastly, it has been observed that when the serum, which has little or no affinity for the red globules in health, readily dissolves them, it is an unerring sign that further bleeding should be avoided. In some of the febrile diseases the fibrine never augments, remains often in normal quantity, and is also often diminished. In the acute inflammatory fever, on the contrary, there is a constant augmentation of this principle, compared with the red globules, as observed by Andral. It is this excess of fibrine which gives firmness to the clot, and is the cause of its being "buffed" and "cupped." The immediate effect of bleeding, according to the same high authority, is to reduce the red globules, but not so with the fibrine; for a reduction of the fibrine does not take place till after a certain time. Such is the state of the blood in the sthenic inflammatory states. There are many reasons, however, for not esteeming the buffed and cupped state of the blood, denoting an excess of fibrine, as a sufficient warranty for bleeding; for these conditions are often present in erysipelas, phthisis, or the early stages of typhus fever; and in either of these cases the loss of a moderate quantity of blood might hurry the patient to his tomb. Again, in acute rheumatism the blood is not only buffed and cupped, but contains a maximum quantity of fibrine; yet the best practitioners seldom think it necessary to take blood, considering that mode of treatment as neither affording present relief nor shortening the course of the disease. The fact, then, of the blood being buffed and cupped does not in all cases warrant venesection. It is also well known that the sthenic or buffed characters of the blood are often greatly modified by the manner in which the blood is drawn; thus, if an individual be bled in both arms, and the blood allowed to flow with different velocities—that is, in a full stream from one and slowly from the other—the blood drawn is identically the same; yet a thick buff will be wanting in the latter and be present in the former. And if the apertures be of different sizes, the same differences will result; the blood from the larger orifice will be buffed, while no such effect is seen in the blood drawn from the smaller one. Again, the form of the vessel which receives the blood, as to whether it be flat or conical, and also its temperature, or whether the blood be received into one that is cold or warm, will also affect the phenomena of its coagulation.

There are many circumstances, therefore, which prevent the blood from being an unerring guide for bleeding in cases of inflammation; but *the assemblage and succession of symptoms* must decide as to the propriety of bloodletting in doubtful cases.

12. An improvement in the character of the secretion or excretion from the inflamed part contraindicates the repetition of bloodletting; for instance, in pneumonia, if the *character* of the expectoration, from being scanty, tenacious, and tinged with blood, becomes



tent of *one-sixth* or more; the fibrine is diminished by *one-third* of its amount; the albumen by *one-seventh*; and, at the same time, it may become loaded with a fetid matter, the product of decomposition (WRIGHT). "Thus it is an agent of terrible activity, and we may well be cautious how we handle it" (HEADLAND). Its sanative power is believed to consist in controlling or preventing the coagulation of lymph; and for this purpose it is used as an auxiliary to bloodletting. It is only useful in the *sthenic* forms of inflammatory action. It is decidedly hurtful in cases of erysipelas disposed to gangrene, in scrofulous states of the system, in debility, and in cases where the nervous system is in an irritable condition, and the condition of the patient tending to the so-called typhoid state. The specific influence of mercury is recognized by the tenderness of the gums which it induces, by the increase in the quantity of saliva, and by the peculiar mercurial feter of the breath. This is the utmost action which should ever be induced. Calomel alone, or calomel combined with opium (a quarter of a grain of the latter with two grains of the former; or a third of a grain of opium with three or four grains of calomel), every three, four, or six hours, is the best form of administration where its influence is rapidly required. But this form of combining mercury with opium should not be persevered in too long, otherwise copious salivation may be induced, which, of all things, ought to be avoided.

Mercury thus employed tends to prevent effusion and favor absorption of effused products. It is advantageously employed in membranous inflammations, and such as go on slowly.

*Antimony* is antiphlogistic, by tending to increase all the secretions; but particularly those from the skin and lungs. It is especially useful in those *sthenic* inflammations which are rapid, and in which a sudden and powerful action is desired; and also where the direct sanative influence of bloodletting is to be maintained. It is thus indispensable in *croup*, extremely efficacious in *sthenic pneumonia*, and highly useful in *bronchitis*. As an agent to keep up the sanative influence obtained by bloodletting, the action of antimony is invaluable; for when bloodletting has weakened the force of the heart by diminishing the pressure on the vessels, then antimony maintains this diminished pressure in proportion as it weakens the force of the heart. A perseverance in its use produces a watery condition of the blood, diminishing especially the amount of fibrine. The production of nausea is an indication that it has taken sufficient effect.

*Alkalies* dissolve the fibrine of the blood and retard its formation; and the ingredients of the urine produced out of the destruction of the albuminous compounds of the body, are increased largely in amount by the administration of alkalies. They generally pass out of the body as salts, having combined with acids in the system, and tending to leave behind them an excess of alkali in the blood.

All treatment ought to be judiciously regulated by the knowledge of the tendency of the disease to a spontaneous favorable termination—the accidental symptoms of urgency requiring treatment and control in many cases, rather than the disease itself.



work *On Acute Diseases* was published, the idea was taken up and acted upon by Franciscus Boissier de Sauvages, a distinguished physician and eminent professor of medicine at Montpellier. He attempted to arrange diseases, as botanists have done plants, into classes, orders, and genera. He endeavored to lay down the characteristic phenomena of each, and to enumerate their principal varieties. The outlines of his nosological system were first published in 1732, and followed thirty years afterwards by his *Nosologia Methodica*—a work which marks an important era in the history of Medicine, as having led to much greater accuracy in the distinction of diseases than was previously observed.

At present the only useful method of defining diseases seems to be an artificial one. It is assumed by nosologists that the proper foundation for the distinction of particular diseases is the occurrence of constant and uniform combinations of morbid phenomena or symptoms, presenting themselves in concourse or in succession. Thus some of the essentials of a definition are obtained, so that each disease may be marked out by such a brief enumeration of its leading characters as might serve to distinguish it from every other. A series of nosological definitions, more or less correct, may be thus provisionally established, so that the same things are designated by the same terms. Objections have been urged to methods of this kind, on the ground that diseases are unsteady and variable in their character; but the aids to science are now so numerous that physicians are becoming more and more able to distinguish diseases from one another, and to tell by what marks, or upon what grounds, they do so distinguish them. Such are the marks or grounds of distinction by which each disease ought to be defined; and as often as we attempt to establish a distinction among diseases, either the deficiencies or the errors of our definitions will be the more easily perceived; and the attempt will lead to a more accurate consideration of observations previously made, as well as to a greater degree of accuracy in subsequent observations. Definitions of diseases are therefore not only of much service to methodical nosology, but they help to render the diagnosis of diseases more perfect. Pathologists, however, are not agreed as to whether the definitions of diseases should be derived from the external phenomena that present themselves in their course, or from the internal pathological conditions on which these phenomena are supposed to depend; and particularly such of these conditions as consist in lesions or structural alterations discoverable after death.

Cullen was in favor of definitions derived from the symptoms; but he believed that the information derived from pathological anatomy might guide to correct distinctions among diseases. Defining diseases by their supposed proximate causes may lead to error, inasmuch as in many cases these causes are disputable, and may long continue to be so. Whatever principle of defining diseases be adopted, it is absolutely necessary that it should be independent of every theoretical view; for any theory employed, however specious, however much we may be persuaded of its truth, may not appear in the same point of view to others, and may therefore occasion end-



mongrel names which pervades medical nomenclature. The idea also of rendering medical nomenclature *uniform*, by deriving the names of diseases from one source only, or from a certain or mixed combination of sources, has caused many to attempt the reform of medical nomenclature, and especially since morbid anatomy has been so much prosecuted that it might serve as a useful guide in distinguishing the disease or dictating its name.

By some it is maintained that "the name of each disease or species should be so characteristic and significant, that a person slightly acquainted with the language and the subject should, on hearing it, immediately understand what is the nature of the disease it designates" (PLOUCQUET). In this respect the name ought to be composed out of the same elements as the definition of the disease—in fact, it ought to be the definition converted into a name, and derived either from the symptoms of the disease or from the supposed proximate cause. But a name which is expressive only of the nature, seat, or proximate cause of a disease may be erroneous in respect of each of these facts singly, or of all of them together. The history of the nomenclature of fever, especially typhoid, would amply illustrate these statements—*e. g.*, putrid fever, adynamic fever, bilious fever, pythogenic fever, enteric fever, meningo-gastric fever, nervous fever, gastric fever, are mild examples of nomenclature and of confusion, which ought to make a man pause before he attempts to construct a new name. It is inexpedient, also, to abandon (except when unavoidable) the names of distinct diseases received and recognized by our forefathers in the science; or of substituting new ones in their place, without an extreme necessity. Sauvages insists much on this point, and Cullen was of the same opinion. "Words," says the former, "are good only in respect of their signification." In dealing, therefore, with ancient nomenclature, which, for the time being, may appear objectionable, it is surely better to extend, if possible, the signification of the word, name, or term, than to alter it. At the same time it must always be permitted to give new names to new diseases, and to select the best out of those which are in use, when a great number have been used to designate one and the same thing. There are some principles, therefore, which it is well to recognize as influencing the judicious choice of a name. Such names, for example, as involve or attempt to indicate a proximate cause are more liable to lead to error than those which are derived from leading symptoms. If names were to be based on supposed causes, new names of diseases would be required whenever a new hypothesis is started. Look, for example, at the names of typhoid fever, already mentioned, and the systems of Linnæus, Vogel, Pinel, and even Mason Good, will show that medical nomenclature has been repeatedly changed without any urgent necessity; and great inconvenience has especially resulted from incorporating particular and often peculiar pathological doctrines with the language and nomenclature of diseases. So much has this been the case, that the language of medical science has been in danger of becoming "a curious mosaic of the chief speculations of ancient and modern times." The pas-





the most valuable information is obtained relative to the health of the people, or of the unwholesomeness and pestilential agencies which surround them. "We can take this or that disease, and measure not only its destructiveness, but its favorite times of visitation; we can identify its haunts and classify its victims." We are able to trace diseases also as they perceptibly get weaker and weaker, or otherwise change their type, as some have done from time to time. We know from the valuable returns of the Registrar-General, prepared periodically by Dr. Farr, that certain diseases are decreasing, or growing less and less destructive; that certain other diseases have ceased in some measure; while other severe diseases have exhibited a tendency to increase. The advantages, therefore, of adopting some system of classifying diseases, which can be put to such useful practical purposes, must be obvious to every one.

To some extent other systems are *natural* in their arrangement, in so far as they attempt to express or exhibit some of the natural relations which subsist among diseases; but the mere expression of one man's interpretation of peculiarities of disease of the same species, and the elevation of such diseases in a classification as specifically distinct, are apt to be based on insufficient evidence as regards natural relations.

**Principles of Classification.**—Many systems of Nosology have been adopted from time to time; and as valuable general principles have been adduced from some, the grounds on which diseases have been classified may be briefly described under the following nine divisions, namely:

I. *The nature of the ascertained causes of disease.* On this principle two classes of diseases are recognized, namely,—(1.) Diseases arising from general causes; (2.) Diseases arising from specific causes.

II. *The pathological states or conditions which attend diseases.* The principle of this classification consists in determining alterations of the structure or the chemical composition of parts, from which names are given to the disease—e. g., pleuritis, pneumonia, &c. The distinctions of Sauvages were generally derived from symptomatic and pathological characters, or external symptoms alone; Cullen, following (1792), adopted similar grounds of classification; but with much more comprehensive views than Sauvages, a more lucid order, and a happier simplicity, he excelled in accuracy of definitions all who had gone before him. His descriptions of disease received no coloring from his theories. They are faithful to nature, consistent with the knowledge of his day; and, greatly in advance of his time, his original and inventive mind dwelt much on the causes of disease in all his reasonings and explanations on medical subjects. Aware, however, of the imperfections of the Art of Medicine, he did not attempt to arrange diseases according to their proximate causes, but according to a method founded partly on their symptoms, partly on their causes, and partly on their seats (CURRIE). A methodical arrangement of this kind has generally been considered the most desirable, as being likely to bring together diseases corresponding not only in some very important relations as regards their symptoms, but also in the indications and means of treatment which they sug-



ment of the orders and subdivisions are determined by the anatomical arrangement of the textures and organs of the animal body, as originally developed by Bichat.

Such is the principle and mode of classification adopted by Dr. Craigie (1836).

VI. A ground of classification exists, having reference to *the general nature and localization of the morbid states*. It comprehends three classes,—(1.) Diseases which occupy the whole system at the same time, and in which all the functions are simultaneously deranged. These have been named general diseases, such as *fevers*. (2.) Constitutional affections, meaning thereby diseases which display themselves in local lesions in any part, or in several parts of the system, but not in all parts at the same time—*e. g.*, *rheumatism*, *gout*. (3.) Local morbid processes.

Such is the classification adopted by Dr. Wood, of Philadelphia (1847).

VII. Applying the principles of a *purely humoral pathology*, we have a classification consisting of,—

*a.* Fevers. *b.* Dyscrasiæ—*e. g.*, *tabes*, *chlorosis*, *scorbutus*, *dropsy*, *diabetes*, *pyæmia*, *tuberculosis*, *carcinoma*. *c.* Constitutional diseases, induced by,—(1.) Specific agents; (2.) Vegetable substances. Such is Wunderlich's arrangement of diseases (1852).

VIII. M. de Savignac, Professor of Clinical Medicine at the Naval School of Toulon, propounded (1861) a Nosological arrangement founded on what he believes to be the “elements” of disease. To each of the classes he so defines, the question would at once suggest itself, and require solution, as to what the “element” may be on which the particular class is made to stand alone. He merely subjoins the word “element” to an adjective formed from the name of each class of diseases. Thus the class Neuroses is distinguished by the *neurotic element*; the class Rheumatalgiæ, by the *rheumatic element*; and so on to the number of fourteen classes. In the formation of orders, genera, or groups of diseases under this classification, no fixed principle can be recognized.

IX. Dr. Stark, of Edinburgh, proposed (1864) an arrangement embracing sixteen classes, namely: (1.) Fevers; (2.) Diseases of the brain, &c.; (3.) Diseases of the heart and organs of circulation; (4.) Diseases of organs of respiration; (5.) Diseases of organs of digestion; (6.) Diseases of urinary organs; (7.) Diseases of organs of generation; (8.) Diseases of organs of locomotion; (9.) Diseases of skin and cellular tissue; (10.) Diseases of uncertain seat; (11.) Malformation; (12.) Debility at birth, and premature birth; (13.) Old age; (14.) Sudden deaths; (15.) Violent or unnatural deaths; (16.) Causes not specified.

None of these nine methods lead to a perfectly philosophical or purely natural classification, because diseases are not yet sufficiently understood to permit us to see clearly their mutual relations; and the best recommendation of any one of them would be a negative one—namely, that of doing the least possible violence to our very imperfect knowledge regarding the natural affinities or alliances of diseases, of which we have at present only a sort of instinctive rec-



tem of classification aims at having the details of its plan to agree in every respect with all the facts as they exist in nature. To effect this end, arrangements, as they *naturally* exist, require to be traced out, not devised. The tracts in which such a pursuit must be followed up, and in which our knowledge is as yet deficient, may be shortly indicated under the following heads, namely: (1.) The affinities or alliances of diseases with each other. (2.) The morbid anatomy of diseased parts. (3.) The communication, propagation, inoculation, generation, development, course, and spontaneous natural termination of diseases. (4.) The connection of the phenomena recognized during life with the facts of morbid anatomy. (5.) The geographical distribution of diseases. (6.) The succession of diseases, so far as they can be traced through past ages; the peculiarities they have exhibited at different periods in the world's history, or within comparatively recent cycles of years. But the time has not yet come for a classification on a basis so comprehensive—simply because the material does not yet exist; and attempts to make so-called *natural* systems of arrangement must end in disappointment, on account of the uncertain and fluctuating data on which they must be based. Such attempts are apt to suggest the serious question, “Whether such Nosology promotes or retards the progress of Medicine?”

**Present State and Aim of Nosology.**—The most distinguished Physicians and Statists are lending their joint aid to obtain a nomenclature and classification of diseases which can be applied to the wants of the civil and military population in every country. Dr. William Farr devised a system of Nosology which was discussed at several meetings of the Statistical Congress of the Great Powers of Europe, convened for the purpose, amongst other business, of devising and adopting a uniform system of nomenclature for recording diseases and the causes of death from them. The Congress met in Paris on the 10th of September, 1855, when a nomenclature of the causes of death was agreed upon, essentially the same as that used in England and Geneva. At a third Conference, held at Vienna in 1857, a *nomenclature* substantially uniform was agreed upon for adoption in all the states of Europe; and fatal cases were to be registered on a uniform plan. Dr. Farr's system of nomenclature has been in use now for many years by the Registrar-General of this country, and more recently by the Army Medical Department. It was also adopted in previous editions of this text-book; and although it had many imperfections and defects, it was still practically the most useful and authoritative Nosology. “In the English list of names,” proposed and adopted by the College of Physicians, “it seemed desirable that as little deviation as possible should be made from those employed by the Registrar-General of England; otherwise his settled plans, and his forms of returns, which have been followed for years, would require to be remodelled; the comparison of future years with past returns would be made difficult and perplexing, if not impossible; and a damaging break would be caused in evidence which becomes more and more



This is a great achievement. The Registrars-General of England, Scotland, and Ireland, the chiefs of the Medical Department of the Army and Navy, and of the British troops in India, have all concurred with others in framing the Nomenclature; and therefore it is not unreasonably expected that greater accuracy, certainty, and uniformity, for comparison, than heretofore will characterize the statistical records of disease, alike in civil life and in the public services. To facilitate the work becoming *international*, the College has translated its nomenclature into Latin, French, German, and Italian equivalents.

The methods of gradually improving the Nomenclature of Diseases in Medicine has some analogy to the methods of gradually improving Representative Reform in Politics. A nomenclature of diseases and a policy of parliamentary representation, judicious and proper a quarter of a century ago, must each eventually give place to the influence of progressive knowledge and power, which invariably come with the rapid movements of the age in which we live. But reform, whether of political representation or medical nomenclature, to be generally acceptable, cannot be the work of one man, nor the accomplishment of a limited period of time. To be on a broad basis and free of prejudice, each work ought to be the combined result of the best men of the time—each man being willing to yield, adapt, and mould his convictions on entering into a mutual arrangement to achieve a common end. Men thus brought together, who differ very seriously as to certain points, may yet, by mutual discussion, come to a definite and reasonable agreement for practical purposes—the terms of the compromise being settled doubtless at the expense of some personal bias, which often has a firmer hold the more imperfect the information of the holder, but which mutual deliberation clears away. Knowing how biassed in opinion individual members of professional and political bodies are apt to be, the unanimous adoption of a Provisional Nomenclature by the

"The German Nomenclature was drawn up by Dr. Hermann Weber, and revised by Dr. Frederic Weber.

"The Italian Nomenclature was drawn up by Dr. Frederic Weber.

"The List of Deformities was drawn up by Dr. Arthur Farre.

"The entire work has been edited by the Secretary, Dr. Sibson; with whom Dr. Barclay took part in editing more especially the Medical portion. The Surgical portions of the Nomenclature were prepared and, in conjunction with the Secretary, edited by Mr. Moore and Mr. Holmes.

"Official changes during the period of the existence of the Committee led also to the introduction into it of the following additional members: Mr. Partridge, as President of the Royal College of Surgeons; Drs. Birkett, Owen Rees, Handfield Jones, Basham, Herbert Davies, Guy, Peacock, Wegg, as Censors of the College of Physicians. Dr. Alderson's first official act after his election as President of the College was to appoint Sir Thomas Watson Chairman of the Committee."

These names are the names of men who hold, or have held, the highest place as representative men in the Science of Medicine; and are at once a guarantee of the intellect and practical knowledge which have been brought to bear upon the work.

The preface to the Nomenclature, from the pen of the Chairman of the Committee, is extremely suggestive; and the work ought to be in the hands of every Student of Medicine, and the handbook of every one who has to do with the Registration of Diseases.





those we now possess, and far less extensive. The nomenclature and classification thus adopted by the College is therefore *strictly provisional*; and it would be well if the Colleges of Physicians and Surgeons in Scotland and in Ireland would unite with that of London in this eminently practical work, and appoint committees to communicate with each other in revising and readjusting such nomenclature at the end of every ten years; and so stamp with their united authority the progressive improvements in the Science of Medicine which are capable of being indicated or expressed in the Nomenclature and Definitions of diseases. Such systematic arrangements, if consistent with existing knowledge, never cramp or hamper a man in carrying out scientific investigations; on the contrary, they enable him to see more clearly in what direction his labor must be advanced, and demonstrate more forcibly than otherwise the deficiencies of his knowledge.

The "Provisional Nomenclature" of the Royal College of Physicians of London, comprehended in the following list, has therefore been adopted in the text of this edition (5th); while the synonyms, equivalents, and definitions have also been incorporated, at the places where the diseases are described in the text, throughout both volumes.

## CHAPTER II.

### TABULAR VIEW OF THE "PROVISIONAL NOMENCLATURE" ADOPTED BY THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

#### GENERAL DISEASES.

A.	b. Anginose. c. Malignant.*
1. Small-pox. <i>Group A</i> (unmodified). <i>Group B</i> (modified). <i>Varieties</i> , applicable to both groups: a. Confluent. b. Semi-confluent. c. Distinct. <i>Syn.</i> , Discrete. d. Abortive. <i>Syn.</i> , Varicelloid. <i>Subordinate Varieties</i> : e. Petechial. f. Hemorrhagic. g. Corymbose. 2. Cow-pox. 3. Chicken-pox. 4. Measles. 5. Scarlet fever. <i>Varieties</i> : a. Simple.	6. Dengue. 7. Typhus fever. 8. Cerebro-spinal fever. <i>Syn.</i> , Malignant purpuric fever; Epidemic cerebro-spinal meningitis. 9. Enteric fever. <i>Syn.</i> , Typhoid fever; and in children is often named Infantile remittent fever.† 10. Relapsing fever. 11. Simple continued fever. 12. Febricula. 13. Yellow fever. 14. Plague. 14 <sup>a</sup> . Beriberi. 15. Ague. <i>Syn.</i> , Intermittent fever. <i>Varieties</i> : a. Quotidian. b. Tertian.

\* Scarlet fever occurs occasionally without any rash or sore throat being observed.

† Fevers symptomatic of worms, teething, or other sources of irritation, should not be included under this head.

- Sub-variety :*  
Double tertian.
- c. Quartan.  
*Sub-variety :*  
Double quartan.
- d. Irregular.
- (100<sup>b</sup>.) *Brow ague.*
16. Remittent fever.\*
17. Simple cholera.
18. Malignant cholera. *Syn.*, Serous cholera; Spasmodic cholera; Asiatic cholera.  
a. Choleraic diarrhœa.
19. Diphtheria.  
a. Diphtheritic paralysis.
20. Whooping-cough.
21. Mumps.
22. Influenza.
23. Glanders.
24. Farcy.
25. Equina mitis. *Syn.*, Grease.
26. Malignant pustule.
27. Phagedæna.
28. Sloughing phagedæna.
29. Hospital gangrene.
30. Erysipelas.  
*Varieties :*  
a. Simple. *Syn.*, Cutaneous.  
b. Phlegmonous. *Syn.*, Cellulocutaneous.  
c. Diffuse inflammation (of cellular tissue).†
31. Pyæmia.‡
32. Puerperal fever.§
33. Puerperal ephemera. *Syn.*, Weed.

- B.
34. Acute rheumatism. *Syn.*, Rheumatic fever.  
(a.) Subacute rheumatism.
35. Gonorrhœal rheumatism.
36. Synovial rheumatism.
37. Muscular rheumatism.  
*Local varieties :*  
a. Lumbago.  
b. Stiff neck.
38. Chronic rheumatism.||
39. Acute gout.
40. Chronic gout.
41. Gouty synovitis.¶
42. Chronic osteo-arthritis. *Syn.*, Chronic rheumatic arthritis.
43. Purpura.  
*Varieties :*  
a. Simple.  
b. Hemorrhagic.
44. Scurvy.  
*Ergotism.*
45. Diabetes. *Syn.*, Diabetes mellitus.
46. Syphilis.  
A. Primary syphilis.  
*Varieties :*  
Hard chancre.  
Indurated bubo.  
Soft chancre.  
Suppurating bubo.  
Phagedænic sore.  
Sloughing sore.  
B. Secondary syphilis.  
c. Hereditary syphilis.
1. \*\*Local syphilitic affections.

\* The malignant local fevers of warm climates are usually of this class.

† In slighter cases, occurring on the surface of the body, this disease is identical with phlegmonous erysipelas.—In registering cases of phlegmonous erysipelas, and of diffuse inflammation arising from injury, surgical operation, or local disease, the cause should be specified.

‡ In returning cases of pyæmia, specify the affected organs.

§ In returning cases of puerperal fever, the more important local lesions, such as peritonitis, effusions into serous and synovial cavities, phlebitis, and diffuse suppuration, should be specified.

|| Cases attended with deposit of lithate of soda are to be returned as chronic gout, and those in which there is marked distortion as chronic osteo-arthritis.

¶ Retrocedent gout is a term applied to cases of gout in which some internal organ becomes affected on the disappearance of the disease from the joints, and should be referred to acute or chronic gout.

\*\* In returning local syphilitic affections, specify whether the case be one of primary syphilis, secondary syphilis, syphilitic deposits, or syphilitic inflammation.—Local syphilitic affections, local cancer, local colloid, and local scrofulous affections, are to be returned in the following order :

Brain.	Supra-renal capsule.	Stomach.	Scrotum.
Spinal cord.	Larynx.	Pylorus.	Testicle.
Nerve.	Bronchi.	Intestines.	Ovary.
Eye.	Lungs.	Rectum.	Fallopian tube.
Eyelid.	Pleura.	Anus.	Uterus.
Orbit.	Mediastinum.	Liver.	Vagina.
Auricle.	Lips.	Hepatic ducts and gall-bladder.	Vulva.
Internal ear.	Mouth.	Pancreas.	Female breast.
Face.	Cheek.	Spleen.	Male mamilla.
Nose.	Jaws.	Peritoneum.	Bone.
Pericardium.	Gum.	Mesenteric glands.	Skull.
Heart.	Tongue.	Kidney.	Joint.
Lymphatics.	Fauces.	Bladder.	Spine.
Lymphatic glands.	Tonsils.	Prostate gland.	Muscle.
Bronchial glands.	Salivary glands.	Penis.	Tendon.
Thyroid gland.	Pharynx.	Prepuce.	Cellular tissue.
Thymus gland.	Œsophagus.		Skin.

47. Cancer. *Syn.*, Malignant disease.\**Varieties:*

- a. Scirrhus. *Syn.*, Hard cancer.
- b. Medullary cancer. *Syn.*, Soft cancer.
- c. Epithelial cancer. *Syn.*, Canceroid epithelioma.
- d. Melanotic cancer. *Syn.*, Melanosis.
- e. Osteoid cancer.†

## 1. Local cancer.‡

48. Colloid. *Syn.*, Colloid cancer; Alveolar cancer.

## 1. Local colloid.§

## NON-MALIGNANT TUMORS AND CYSTS.||

Fibrous tumor.

Fibro-cellular tumor.¶

Fibro-nucleated tumor.

Fibro-plastic tumor.\*\*

Myeloid tumor.

Fatty tumor. *Syn.*, Lipoma.

Osseous tumor.

- a. Of bone. *Syn.*, Exostosis.

*Varieties:* Ivory.

Cancelled.

Diffused.

- b. Of the soft parts.

Cartilaginous tumor. *Syn.*, Enchondroma.

Fibro-cartilaginous tumor.

Glandular tumor. *Syn.*, Adenocèle.

Vascular tumor.

Nævus.

Sebaceous tumor.

Cholesteotoma.

Molluscum.

Warty tumor and warts.

Condyloma.

Cheloid.

Villous tumor.

Simple or barren cysts.

a. Serous.

b. Synovial. *Syn.*, Bursal.

c. Mucous.

d. Suppurating.

e. Sanguineous.

f. Hemorrhagic.

g. Aneurismal.

h. Oily.

i. Colloid or gelatinous.

j. Seminal.

Compound or proliferous cysts.

- a. Complex cystic tumor. *Syn.*, Cysto-sarcoma. With intracystic growths.

- b. Cutaneous or piliferous. *Syn.*, Dermoid.

- c. Dentigerous.

## 49. Lupus.

*Varieties:*

- a. Chronic lupus.

- b. Lupus exedens.

## 50. Rodent ulcer.

51. True leprosy. *Syn.*, Elephantiasis Græcorum.

## 52. Scrofula.

*Varieties:*

- a. Scrofula with tubercle.

- b. Scrofula without tubercle.††

## 1. Local scrofulous affections.

Tubercular meningitis.

Scrofulous ophthalmia.

Scrofulous iritis.

Tubercular pericarditis.

Phthisis pulmonalis.

Scrofulous disease of glands.

Acute miliary tuberculosis.

Tabes mesenterica.

Tubercular peritonitis.‡‡

## 53. Rickets.

## 54. Cretinism.

*Varieties:*

- a. Complete cretinism. *Syn.*, Incurable cretinism.

\* In returning cases of cancer in more than one organ, specify in which the disease is primary, and in which secondary.—State also the kind and duration of the disease in each case, and the nature of all operations, with their dates and results.

† Cancer in mucous membranes, when covered by a villous growth, has received the name of Villous cancer.

‡ In returning cases of local cancer, specify the variety of cancer, by adding, after "46," the letter a, b, c, d, or e, according to the nature of the case. They are to be returned in the order specified in the foot-note (\*\*) on preceding page.

§ Cases of local colloid are to be returned in the order specified in the foot-note (\*\*) on preceding page.

|| In order that the malignant and non-malignant growths may appear together, the non-malignant tumors and cysts are inserted here. They should, however, be returned under "Non-malignant tumors" among the local diseases, and they are not, therefore, numbered at this place.

¶ When occurring as a pendulous outgrowth from a mucous surface, it constitutes the chief varieties of Polypus.

\*\* When the fibro-cellular or fibro-plastic tumor, but more especially the latter, slowly involves the adjacent soft structures, and returns after removal, it has received the name of Recurrent fibroid.

†† The constitutional tendency which has received the name of the Scrofulous Diathesis, when unattended by local lesions, is not to be returned under the heading of Scrofula.

‡‡ These and all other cases of local scrofulous affection are to be returned in the order specified in the foot-note (\*\*) on preceding page.

- b. Demi-cretinism.  
c. Incomplete cretinism. *Syn.*, Curable cretinism.

55. \*Anæmia.  
56. Chlorosis. *Syn.*, Green-sickness.  
57. \*General dropsy.†

## DISEASES OF THE NERVOUS SYSTEM.‡

### DISEASES OF THE BRAIN AND ITS MEMBRANES.

58. Encephalitis.§  
59. Meningitis.  
1. Inflammation of the dura mater.||  
2. Inflammation of the pia mater and arachnoid.  
(58<sup>1</sup>). 3. *Tubercular meningitis.* *Syn.*, *Acute hydrocephalus.*  
60. Inflammation of the brain.  
61. Red softening (of the brain).  
62. Yellow softening (of the brain).  
63. Abscess (of the brain).  
64. Apoplexy.  
*Varieties:*  
a. Congestive.  
b. Sanguineous. *Syn.*, Cerebral hemorrhage.  
65. Sunstroke.  
66. Chronic hydrocephalus.  
67. Hypertrophy (of the brain).  
68. Atrophy (of the brain).  
69. White softening (of the brain).  
*Syn.*, Atrophic softening.¶  
(48<sup>1</sup>). *Syphilitic disease.*

- (49<sup>1</sup>). *Cancer.*  
70. Fibrous tumor.  
71. Osseous tumor.  
(53<sup>1</sup>). *Tubercular deposit.*  
a. *Miliary or granular tubercle.\*\**  
b. *Yellow tubercle.*  
72. Parasitic disease.  
73. Diseases of the cerebral arteries.  
1. Degeneration (fatty and calcareous). *Syn.*, Atheroma. Ossification.  
2. Aneurism.  
3. Impaction of coagula.  
1. Thrombosis (local coagulation).  
2. Embolism (coagula conveyed to a distance).

### DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

74. Inflammation.††  
*Varieties:*  
a. Spinal meningitis.  
b. Myelitis.  
75. Hemorrhage (spinal). *Syn.*, Spinal apoplexy.

\* When the cause of this affection has been ascertained, the case should be returned under the head of the Primary disease, the secondary affection being also specified.

† Local dropsies, such as ovarian, and effusions into the serous cavities, as hydrothorax or ascites, when not connected with anasarca, should be returned as local diseases.

### ‡ ARRANGEMENT OF LOCAL DISEASES.

The Local Diseases have been drawn up in accordance with the following arrangement :

Catarrh.	Passive congestion.	Atrophy.	Cyst.
Inflammation.	Extravasation of	Degeneration.	<i>Scrofula.</i>
Ulcerative inflamm'n.	blood.	Fatty and calcareous. <i>Syn.</i> , Atheromatous.	a. <i>With tubercle.</i>
Suppurative "	Hemorrhage.	Fibroid.	b. <i>Without tubercle.</i>
Plastic "	Dropsy.	Lardaceous disease.	Parasitic disease.
<i>Pyæmic</i> "	Fibrinous deposit.	<i>Syphilitic disease.</i>	Calculus and concretions.
Rheumatic "	Alteration of dimensions.	<i>Cancer.</i>	Malformation.
Gouty "	Dilatation.	<i>Colloid.</i>	<i>Injury.</i>
<i>Syphilitic</i> "	Contraction.	Non-malignant tumor.	<i>Foreign body.</i>
<i>Scrofulous</i> "	Hypertrophy.		Functional diseases.
Gonorrhœal "			
Gangrene.			

The diseases printed in italics are to be returned, not among the local diseases, but under the heading referred to by number.

§ This term is to be used only when the precise seat of the inflammation has not been ascertained by post-mortem examination.

|| This form of inflammation is almost invariably the result of injury or disease of the bones of the skull; in such cases, the injury or disease by which it is caused ought to be specified.

¶ This form of disease is the result of imperfect nutrition, owing to deficient supply of blood, and is in most instances dependent upon mechanical obstruction, or degeneration of the cerebral arteries.

\*\* To be referred to tubercular meningitis.

†† This term is to be used only when the precise seat of the inflammation has not been ascertained by post-mortem examination.

76. Atrophy (spinal). *Syn.*, Tabes dorsalis.  
 77. White softening (spinal).  
 78. Spina bifida.  
 (49<sup>1</sup>). Cancer.  
 79. Non-malignant tumor.

## DISEASES OF NERVES.

80. Inflammation.  
 81. Atrophy.  
 (49.) Cancer.  
 82. Neuroma.  
 83. \*Paralysis.  
 (107.) 1. *Paralysis of the insane.* *Syn.*,  
*General paralysis.*  
 84. 2. \*Hemiplegia.  
 85. 3. \*Paraplegia.  
 86. Locomotor ataxy.  
 (79<sup>1</sup>.) *Progressive muscular atrophy.*  
 87. 5. Infantile paralysis.  
 6. \*Local paralysis.†  
 a. Scrivener's palsy.  
 7. *Diphtheritic paralysis.*  
 8. *Lead palsy.*  
 Paralysis from Lathyrus (sativa).

## GENERAL DISEASES OF THE NERVOUS SYSTEM.

88. Tetanus.  
 89. Hydrophobia.  
 90. Infantile convulsions.  
 91. Epilepsy.  
 Epileptic vertigo. *Syn.*, Petit mal.  
 92. \*Convulsions.

93. Spasm of muscle.  
 94. Laryngismus stridulus. *Syn.*, Spasm of the glottis. Spasmodic croup. Child-crowing.  
 95. Shaking palsy.  
 96. *Mercurial tremor.*  
 97. Chorea. *Syn.*, St. Vitus's dance.  
 a. Acute.  
 b. Chronic.  
 98. Hysteria.  
 99. Catalepsy.  
 100. Neuralgia.  
*Principal varieties:*  
 a. Facial. *Syn.*, Tic douloureux.  
 b. Brow ague. *Syn.*, Hemicrania.  
 c. Sciatica.  
 101. \*Hyperæsthesia.  
 102. \*Anæsthesia.  
 103. *Delirium tremens.*

## DISORDERS OF THE INTELLECT, &amp;c.

104. Mania.  
 a. Acute mania.  
 b. Chronic mania.  
 105. Melancholia.‡  
 106. Dementia.  
 a. Acute dementia.  
 b. Chronic dementia.  
 107. Paralysis of the insane. *Syn.*, General paralysis.  
 108. Idiocy. (Congenital.)  
 109. Imbecility. (Congenital.)  
 110. Hypochondriasis.

## DISEASES AND INJURIES OF THE EYE.

## CONJUNCTIVA.

111. Conjunctivitis. *Syn.*, Ophthalmia.  
 112. Catarrhal ophthalmia.  
 113. Pustular ophthalmia.  
 114. Purulent ophthalmia.  
 115. Purulent ophthalmia of infants.  
*Syn.*, Ophthalmia neonatorum.  
 (53<sup>1</sup>.) *Scrofulous ophthalmia.* *Syn.*, *Strumous ophthalmia.*  
 116. Exanthematous ophthalmia.  
 117. Gonorrhœal ophthalmia.  
 118. Chronic ophthalmia.  
 119. Œdema of the sub-conjunctival tissue. *Syn.*, Chemosis.  
 120. Pinguecula.  
 121. Pterygium.  
 122. Fatty tumors.  
 123. Cysticercus telæ cellulossæ.  
 124. Metallic stains.  
 a. From nitrate of silver.  
 b. From lead.

## CORNEA.

125. Keratitis.  
 126. Chronic interstitial keratitis.  
 127. Keratitis with suppuration. *Syn.*, Onyx.  
 128. Ulcer.  
 129. Opacity. *Syn.*, Leucoma.  
 130. Conical cornea.  
 131. Arcus senilis.  
 132. Staphyloma.  
 133. Entozoa in the anterior chamber.

## SCLEROTIC.

134. Scleritis.  
 135. Staphyloma.

## IRIS.

136. Iritis.  
 137. Traumatic iritis.  
 138. Rheumatic iritis.  
 139. Arthritic iritis.

\* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When the cause of any of these forms of paralysis has been ascertained, it should be stated.

‡ Cases of so-called monomania to be classed under chronic mania or melancholia, according to their character.

- (40<sup>1</sup>.) *Syphilitic iritis.*  
 (58<sup>1</sup>.) *Scrofulous iritis.*  
 140. Gonorrhœal iritis.  
 141. Sequelæ of iritis.  
 142. Congenital defects of iris.

## CHOROID AND RETINA.

143. Choroiditis.  
 144. Retinitis.  
 145. Choroidal apoplexy.  
 146. Amaurosis.  
 147. Impaired vision.  
 148. *Muscæ volitantes.*  
 149. Albinism.

## VITREOUS BODY.

150. Synchysis.  
 151. Various morbid deposits.  
 152. Entozoa.

## LENS AND ITS CAPSULE.

153. Cataract.  
*Varieties:*  
   *a. Hard.*  
   *b. Soft.*  
   *c. Fluid.*  
 154. Congenital cataract.  
 155. Traumatic cataract.

## GENERAL AFFECTIONS OF THE EYE.

156. Glaucoma.  
 157. Hydrophthalmia.  
 (49<sup>1</sup>.) *Cancer.*  
 (58.) *Scrofulous deposit within the eyeball.*  
 158. Total disorganization of the eye from injury.

## VARIOUS DEFECTS OF SIGHT.

159. Short sight.  
 160. Long sight.  
 161. Faulty perception of colors. *Syn.,*  
   Color blindness.

162. Hemeralopia.  
 163. Nyctalopia.  
 164. Astigmatism.

## DISEASES OF THE LACHRYMAL APPARATUS.

165. Lachrymal obstruction.  
 166. Abscess and fistula.  
 167. Dacryolith.  
 168. Diseases of the lachrymal gland and its ducts.

## DISEASES OF THE EYELIDS.

169. Inflammation.  
 170. Hordeolum.  
 171. Abscess in the Meibomian glands.  
 172. Epicanthis  
 173. Entropium.  
 174. Ectropium.  
 175. Trichiasis.  
 176. Madarosis. *Syn.,* Loss of the eye-lashes.  
 177. Tarsal ophthalmia.  
 178. Blepharospasmus.  
 (49.) *Cancer.*  
 179. Cyst of the lids.  
   *Phthiriasis.*

## DISEASES OF THE PARTS WITHIN THE ORBITS.

180. Abscess in the orbit.  
 181. Strabismus.  
 182. Protrusion of the eyeball. *Syn.,*  
   *Proptosis.*  
 (277.) *Ex-ophthalmic bronchocele.*  
 (248.) *Orbital aneurism.*  
 (49.) *Cancer.*  
 183. Non-malignant tumor.  
 184. Hydatid tumor in the orbit.  
 185. Affections of orbital nerves.  
   (*Injuries of the eye are given at p.*  
   197.)\*

## DISEASES OF THE EAR.

## AURICLE.

186. Gouty and other deposits.  
 187. Hæmatoma auris.  
 (49<sup>1</sup>.) *Cancers.*  
 188. Non-malignant tumor.  
 (821, &c.) *Cutaneous affections.*  
 189. Malformations.  
 (1007.) *Injuries.*

## EXTERNAL MEATUS.

190. Inflammation.  
   *a. Acute.*  
   *b. Chronic.*  
 191. Abscess.  
 192. Accumulation of wax.  
 193. Polypus.  
 194. Sebaceous tumor. *Syn.,* Molluscous.

195. Exostosis.  
 (1007.) *Foreign bodies.*

## MEMBRANA TYMPANI.

196. Inflammation.  
 197. Ulceration.  
 198. Perforation.  
 (1007.) *Injuries.*

## EUSTACHIAN TUBE.

199. Obstruction.

## TYMPANUM.

200. Disease of the mucous membrane.  
 201. " " ossicles.  
 202. " " mastoid cells.

\* When any of these affections implicate the brain, carotid artery, or lateral sinus, the fact should be stated.

## INTERNAL EAR.

203. Organic disease.  
 204. Necrosis of petrous bone.  
 205. Deafness.

- a. Functional or nervous.  
 b. From disease.  
 c. Deaf-dumbness.  
 (49<sup>1</sup>.) *Cancer*.\*

## DISEASES OF THE NOSE.

206. Hypertrophy. *Syn.*, Lipoma.  
 207. Wart.  
 208. Sebaceous cyst.  
 (49<sup>1</sup>.) *Cancer of the skin*.  
 (51<sup>1</sup>.) *Lupus*.  
 209. Ozena.  
 210. Abscess of the septum.  
 210<sup>a</sup>. Ulceration of the pituitary membrane.  
 211. Perforation of the septum.  
 212. Epistaxis.  
 213. Hypertrophy of the pituitary membrane.

214. Polypus nasi.  
*Varieties*:  
 a. Gelatinous.  
 b. Fibrous.  
 Naso-pharyngeal polypus.  
 215. Non-malignant tumors of the septum.  
 216. Rhinoliths.  
 (1008.) *Foreign bodies*.  
 (49<sup>1</sup>.) *Cancer*. *Syn.*, *Malignant polypus*.  
 Loss or perversion of sense of smell.

## DISEASES OF THE CIRCULATORY SYSTEM.

## DISEASES OF THE HEART AND ITS MEMBRANES.

## DISEASES OF THE PERICARDIUM.

- 216<sup>a</sup>. Pericarditis.  
 217. Suppurative pericarditis.  
 (53<sup>1</sup>.) *Tubercular pericarditis*.  
 218. Adherent pericardium.†  
 219. Dropsy.  
 (49<sup>1</sup>.) *Cancer*.  
 220. Malformations.

## DISEASES OF THE ENDOCARDIUM.

221. Endocarditis.‡  
 222. Valve-disease.  
 1. Aortic.  
 2. Mitral.  
 3. Of pulmonary artery.  
 4. Tricuspid.  
*Varieties*:  
 a. Vegetations.  
 b. Fibroid thickening.  
 c. Atheromatous and calcareous degeneration.  
 d. Aneurism.  
 e. Laceration.  
 f. Simple dilatation of orifice.  
 g. Malformations.  
 Obstruction to the circulation, or Regurgitations should be specially noted when they accompany the valve-disease.  
 223. Fibrinous concretions in the cavities of the heart.§

## DISEASES OF THE MUSCULAR STRUCTURE OF THE HEART.

223. Myocarditis.  
 224. Abscess.||  
 225. Hypertrophy.  
 a. Of left side.  
 b. Of right side.  
 226. Dilatation.  
 a. Of left side.  
 b. Of right side.  
 227. Atrophy.  
 228. Excess of fat.  
 229. Fatty degeneration.  
 230. Fibroid degeneration.  
 231. Aneurism.  
 232. Acute aneurism.  
 233. Rupture.¶  
 (49<sup>1</sup>.) *Cancer*.  
 234. Entozoa.  
 235. Disease of the coronary arteries.  
 236. Malformations.\*\*  
 237. Cyanosis.  
 238. *Injuries of the heart*.  
 239. †† Angina pectoris.  
 240. †† Syncope.  
 241. †† Palpitation and irregularity of the heart.

## DISEASES OF THE BLOODVESSELS.‡‡

## DISEASES OF THE ARTERIES.

242. Arteritis.  
 243. Degeneration. Fatty and calcareous.  
*Syn.*, Atheroma. Ossification.

\* When any of these affections implicate the brain, carotid artery, or lateral sinus, the fact should be stated.

† Including under this term partial adhesions and calcareous and ossific deposits.

‡ In returning such cases state, if possible, the valve or valves affected.

§ Cases are to be returned under this head only when the condition has evidently existed during life, and is believed to have been the cause of death.

¶ Abscess dependent on pyæmia should be referred to that disease.

¶ In returning cases of aneurism and rupture, the situation ought to be stated.

\*\* State which, according to list, page 201.

†† When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

‡‡ The vessel affected should in all cases be specified.



244. Narrowing and obliteration.  
 246. Occlusion.  
   *a.* From compression.  
   *b.* From impaction.  
     1. Thrombosis (local coagulation).  
     2. Embolism (coagula conveyed from a distance).  
 247. Dilatation.  
 248. Aneurism.  
   In returning such cases, state whether the aneurism be—  
   *a.* Fusiform.  
   *b.* Saccular, or  
   *c.* Diffused (sac formed by the surrounding tissue).  
 249. Rupture of artery.  
   *a.* From disease of artery.  
   *b.* From disease external to artery.  
 250. Partial rupture of artery. *Syn.*, Dissecting aneurism.  
 251. Traumatic.  
 252. Arterio-venous aneurism.  
 253. Aneurismal varix.  
   *Varieties:*  
     *a.* Traumatic.  
     *b.* Spontaneous.  
 254. Varicose aneurism.  
   *Varieties:*  
     *a.* Traumatic.  
     *b.* Spontaneous.  
 255. Cirroid aneurism. *Syn.*, Arterial varix.

256. Aneurism by anastomosis.  
 257. Malformation.  
   *a.* Constriction or occlusion of the commencement of the descending aorta (originating in partial malformation).  
 (1049<sup>a</sup>.) *Injuries of arteries.*  
 (1049<sup>a1</sup>.) *Contusion.*  
 (1049<sup>a2</sup>.) *Laceration. Syn., Partial rupture.*  
     *a.* Of outer coat.  
     *b.* Of inner coat.  
   *Wound.*

## DISEASES OF VEINS.

- 257<sup>a</sup>. Phlebitis.  
   *Varieties:*  
     *a.* Adhesive.  
       Phlegmasia dolens.  
     *b.* Suppurative.  
 258. Phlegmasia dolens.  
 258. Fibrinous concretions in the veins.  
 259. Obstruction.  
 260. Obliteration.  
 261. Phlebolithes.  
 262. Varicose veins.  
 263. Nævus vascularis.  
 (1049<sup>b</sup>.) *Injuries of veins.*  
 (1049<sup>b1</sup>.) *Rupture, without external wound.*  
 (1049<sup>b2</sup>.) *Wound of vein with entrance of air.*

## DISEASES OF THE ABSORBENT SYSTEM.

264. Inflammation of lymphatics.  
 265. Suppuration of lymphatics.  
 266. Inflammation of glands.  
 267. Suppuration of glands.  
 268. Hypertrophy of glands.  
   *a.* Chronic enlargement of glands.  
 269. Atrophy of glands.  
 270. Lymphatic fistula.  
 (1182.) *Foreign bodies and concretions.*  
 271. Obstruction of the thoracic duct.\*  
 272. Obstruction, obliteration, and varicosity of lymphatics.  
 278. Bursting of lymphatics.  
 (49.) *Syphilitic bubo.*

- (49.) *Syphilitic inflammation of glands.*  
 (50.) *Cancer.*  
 (53.) *Scrofulous disease of glands.*  
   *Suppuration.*  
 (1183.) *Wounds of lymphatics.*

*Diseases of the Bronchial Glands.*

- (338.) *Inflammation.*  
 (339.) *Abscess.*  
 (340.) *Enlargement.*  
 (49<sup>1</sup>.) *Cancer.*  
 (341.) *Non-malignant growth.*  
 (53<sup>1</sup>.) *Tubercle.*

## DISEASES OF DUCTLESS GLANDS.

## DISEASES OF THE THYROID GLAND

274. Inflammation.  
   *a.* Acute.  
   *b.* Chronic.  
 275. Goitre.  
 276. Cyst.  
 277. Exophthalmic bronchocele.  
 278. Pulsating bronchocele.  
 (49<sup>1</sup>.) *Cancer.*

## DISEASES OF THE THYMUS GLAND.

279. Hypertrophy.

- (50.) *Cancer.*  
 280. Non-malignant tumor.

## DISEASES OF THE SUPRA-RENAL CAPSULES.

281. Suppuration.  
 (49<sup>1</sup>.) *Cancer.*  
 (53.) *Tubercular degeneration.*  
 282. Addison's disease. *Syn.*, Bronze skin. *Melasma Addisoni.*

\* The cause of the obstruction should be stated.

## DISEASES OF THE RESPIRATORY SYSTEM.

## DISEASES OF THE RESPIRATORY SYSTEM NOT STRICTLY LOCAL.

283. Hay asthma.  
 (22.) *Influenza*.  
 (20.) *Whooping-cough*.  
 284. Croup.  
 (19.) *Diphtheria*.  
 (989.) \**Asphyxia*.

## DISEASES OF THE NOSTRILS.

285. Coryza. *Syn.*, Nasal catarrh.

## DISEASES OF THE LARYNX.

290. Inflammation of the epiglottis.  
 291. Ulceration of the epiglottis.  
 292. Laryngeal catarrh.  
 293. Laryngitis.  
     *a.* Acute.  
     *b.* Chronic.  
 294. Ulcer.†  
 295. Abscess.  
 296. Œdema of the glottis.  
 297. Necrosis of cartilage (see the previous Note).  
 298. Contraction.  
 (49<sup>1</sup>.) *Epithelial cancer*.  
 299. Warty growth.  
 300. Polypus.  
 301. Cyst.  
 (1036.) *Foreign bodies in the larynx*.

## FUNCTIONAL AFFECTIONS OF THE LARYNX.

302. \*Aphonia.  
 303. \*Paralysis of the glottis.  
 304. \*Spasm of the glottis.  
 (94.) *Laryngismus stridulus*.

## DISEASES OF THE TRACHEA AND BRONCHI.

305. Bronchial catarrh.  
 306. Bronchitis.  
     *a.* Acute.  
     *b.* Chronic.  
 307. Ulcer.  
 308. \*Casts of the bronchial tubes.  
 309. Necrosis of the cartilages of the trachea.‡  
 310. Dilatation.  
 311. Contraction.  
 (49<sup>1</sup>.) *Cancer*.

312. Non-malignant Tumor.  
 (53<sup>1</sup>.) *Tubercle*  
 (1036.) *Foreign body in the bronchi*.  
 313. Asthma.

## DISEASES OF THE LUNG.

314. Pneumonia.  
     *Variety:*  
     *a.* Lobular.§  
 315. Abscess.  
 (28.) *Pyæmic inflammation and abscess*.  
 316. Gangrene.  
 317. \*Passive congestion.  
 318. \*Pulmonary extravasation.  
     Pulmonary apoplexy.  
 319. \*Œdema.  
 320. Cirrhosis.  
 321. Emphysema.  
     *a.* Vesicular.  
     \**b.* Interlobular.  
 322. Atelectasis.  
 323. \*Collapse.  
 (48<sup>1</sup>.) *Syphilitic deposit*.  
 (49<sup>1</sup>.) *Cancer*.  
 (53<sup>1</sup>.) *Phthisis*.  
 (53<sup>1</sup>.) *Acute miliary tuberculosis*.  
 324. Acute pneumonic phthisis.  
 325. Hydatid.  
 325\*. Chronic pneumonic phthisis.  
 (1042-46.) *Injuries*.  
 (1036.) *Foreign bodies*.  
 326. Millstone makers' phthisis.  
 327. Grinders' asthma.  
 328. Miners' asthma.

## DISEASES OF THE PLEURA.

329. Pleurisy.  
 330. Chronic pleurisy.  
 331. Empyema.  
 332. Adhesions, including thickening and ossification.  
 333. \*Hydrothorax.  
 334. Pneumothorax.  
 (49<sup>1</sup>.) *Cancer*.  
 335. Non-malignant tumor.  
 (53<sup>1</sup>.) *Tubercular pleurisy*.  
 (1045.) *Injuries*.

## DISEASES OF THE MEDIASTINUM.

336. Abscess.  
 (49<sup>1</sup>.) *Cancer*.  
 337. Non-malignant tumor.  
 (279.) *Diseases of the thymus gland*.

\* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When chronic laryngitis, ulcer of the larynx, or necrosis of the cartilage (*see below*), is due to phthisis or syphilis, the terms *Syphilitic* or *Phthisical* should be prefixed to the designation of the disease, and the case ought to be returned under the head of the primary affection.

‡ When this affection is due to phthisis or syphilis, the terms *Syphilitic* or *Phthisical* should be prefixed to the designation of the disease, and the case ought to be returned under the head of the primary affection.

§ The term Secondary has been applied to pneumonia when it occurs as a complication of some other disease; such cases ought to be returned under the head of the primary affection.

## DISEASES OF THE BRONCHIAL GLANDS.

388. Inflammation.  
389. Abscess.  
340. Enlargement.

(49<sup>1</sup>.) *Cancer*.

841. Non-malignant tumor.

(58<sup>1</sup>.) *Tubercle*.

## DISEASES OF THE DIGESTIVE SYSTEM.

## DISEASES OF THE LIPS.

*The affected lip ought to be specified.*

342. Ulcer.  
(48<sup>1</sup>.) *Syphilitic ulcer*.  
343. Fissures.  
(49<sup>1</sup>.) *Cancer*.  
(58<sup>1</sup>.) *Strumous hypertrophy*.  
344. Cyst.  
345. Malformations.  
346. Hare-lip.

## DISEASES OF THE MOUTH.

347. Stomatitis.  
348. Ulcerative stomatitis.  
349. Thrush. *Syn.*, Aphtha. Vesicular stomatitis.  
350. Parasitic thrush. *Syn.*, Parasitic aphthæ.  
351. Abscess of the cheek.  
352. Cancrum oris. *Syn.*, Gangrenous stomatitis.  
353. Cysts of the cheek.  
354. Ranula.  
(49<sup>1</sup>.) *Cancer*.

## DISEASES OF THE JAWS (exclusive of the Alveoli).

355. Adhesion of the jaws by cicatrix.  
356. Abscess of the antrum.  
(49<sup>1</sup>.) *Cancer*.  
357. Fibrous tumor.  
358. Myeloid tumor.  
359. Osseous tumor.  
Hypertrophy of the bones of the face.  
360. Cartilaginous tumor.  
361. Vascular tumor.  
362. Cyst.

## DISEASES, MALFORMATIONS, AND INJURIES OF THE TEETH, GUMS, AND ALVEOLI.

368. Teething.\*

## DISEASES OF THE DENTAL TISSUE.

364. Caries.  
365. Necrosis.  
366. Exostosis.  
367. Absorption.

## DISEASES OF THE DENTAL PULP.

368. Irritation.

869. Inflammation.  
870. Ulceration.  
871. Gangrene.  
872. Granulation or polypus.  
873. Calcification.

## DISEASES OF THE DENTAL PERIOSTEUM.

874. Inflammation.  
875. Gum-boil.  
876. Chronic thickening.  
877. Rheumatic inflammation.

## DISEASES OF THE GUMS.

878. Inflammation.  
879. Ulceration.  
880. Hypertrophy.  
881. Atrophy.  
882. Induration (in infancy).  
(50<sup>1</sup>.) *Cancer*.  
883. Non-malignant tumor.  
a. Polypus.  
b. Cartilaginous tumor.  
c. Vascular tumor.  
884. Epulis.

## DISEASES OF THE ALVEOLI.

885. Inflammation.  
886. Necrosis.  
887. Caries.  
888. Exostosis.  
889. Dentigerous cyst.  
890. Absorption.

## SPECIFIC DISEASES AFFECTING THE DENTAL PERIOSTEUM, GUMS, OR ALVEOLI.

891. Mercurial inflammation.  
892. Phosphoric inflammation and necrosis.  
(902<sup>c</sup>.) *Blue gum from lead*.  
(45.) *Scurvy*.

## IRREGULAR DENTITION.

- Irregularity in the time of eruption of the—  
893. Temporary teeth.  
894. Permanent teeth.  
Irregularity in the position of the—  
895. Temporary teeth.  
896. Permanent teeth.  
Irregularity of the number of the—  
897. Temporary teeth.  
898. Permanent teeth.  
Irregularity in the form of the—

\* Any affection, such as convulsions and paralysis, induced by this condition, should be specified.

399. Temporary teeth.  
 400. Permanent teeth.  
 Abnormal development of the—  
 401. Dental tissue.  
 402. Enamel.  
 403. Dentine.  
 404. Cementum.  
 405. Alveolar portions of the jaws, in size.  
 406. Alveolar portions of the jaws, in form.  
 407. Defective growth of lower jaw.  
 408. Mechanical injuries of the alveoli and dental periosteum.  
   *a.* Hemorrhage.  
   *b.* Fracture.  
 409. Mechanical injuries of the teeth.  
   *a.* Fracture.  
   *b.* Dilaceration.  
   *c.* Dislocation.  
   *d.* Friction.

## DISEASES OF THE TONGUE.

410. Glossitis.  
 411. Ulceration.  
 412. Aphthous ulcer.  
 413. Abscess.  
 414. Hypertrophy.  
 (48<sup>1</sup>.) *Primary syphilis.*  
 (48<sup>1</sup>.) *Secondary syphilis.*  
 (49<sup>c</sup>.) *Epithelial cancer.*  
 415. Vascular tumor.  
 416. Tongue-tie.  
 (87.) *\*Paralysis.*

## DISEASES OF THE FAUCES AND PALATE.

417. Sore throat.  
 418. Relaxed throat.  
 419. Ulcerated throat.  
 420. Quinsy. *Syn.*, Cynanche tonsillaris.  
 421. Tonsillitis.  
 422. Sloughing sore throat. *Syn.*, Putrid sore throat. Cynanche maligna.†  
 (19.) *Diphtheria.*  
 423. Enlarged tonsils.  
 (49<sup>1</sup>.) *Cancer of tonsils.*  
 (53<sup>1</sup>.) *Scrofulous diseases of tonsils.*  
 424. Elongated uvula.  
 425. Perforation of the palate.  
 426. Stricture of the fauces.  
 (48<sup>1</sup>.) *Syphilitic affection of fauces and tonsils.*  
 (49<sup>1</sup>.) *Cancer.*  
 427. Non-malignant tumor.  
   *a.* Fibro-cellular tumor.  
   *b.* Fibro-cystic tumor.  
 428. Cleft palate.

## DISEASES OF THE PHARYNX.

429. Pharyngitis.  
 430. Ulcer.  
   *a.* Superficial ulcer.  
   *b.* Perforating ulcer.  
 431. Abscess.  
 432. Sloughing.  
 433. Adhesion of soft palate.  
 434. \*Dilatation.  
 (48<sup>1</sup>.) *Syphilitic affection.*  
 (49<sup>1</sup>.) *Cancer.*  
 (1037.) *Foreign bodies.*  
 (87.) *\*Paralysis.*

## DISEASES OF THE SALIVARY GLANDS.

435. Inflammation.  
 436. \*Salivation. *Syn.*, Ptyalism.  
 437. Abscess.  
 438. Salivary fistula.  
 (21.) *Mumps.*  
 (49<sup>1</sup>.) *Cancer.*  
 439. Non-malignant tumor.  
 440. Salivary calculus.‡

## DISEASES OF THE ŒSOPHAGUS.

441. Œsophagitis.  
 442. Ulceration.  
 443. \*Perforation.  
 444. \*Stricture.  
 (49<sup>1</sup>.) *Cancer.*  
 (1038.) *Foreign bodies.*  
 445. Malformations.  
 (83.) *\*Paralysis.*  
 446. Dysphagia.

## DISEASES OF THE STOMACH.

447. Gastritis.  
 (916, &c.) *a. From irritant poisons.*  
 448. Chronic ulcer.  
 449. Hæmatemesis.  
 450. Perforation.§  
 451. \*Dilatation.  
 452. \*Stricture.  
 453. Gastric fistula.  
 454. Hernia.  
 (49<sup>1</sup>.) *Cancer.*  
 (50<sup>1</sup>.) *Colloid.*  
 455. Non-malignant tumor.  
 456. Sarcinæ.  
 (1061-63.) *Injuries to the stomach.*  
 (1065.) *Foreign bodies.*  
 457. Laceration (spontaneous).  
 458. Dyspepsia.  
 459. Gastrodynia.  
 460. Pyrosis.  
 461. \*Vomiting.

\* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† This affection must be distinguished from malignant scarlet fever.

‡ Whenever any of the affections of the mouth, throat, or parts connected therewith depend on syphilis, scurvy, local irritants, or any other specific cause, the fact should be stated.

§ The cause of the perforation, when ascertained, should be stated.

## DISEASES OF THE INTESTINES.

462. Enteritis.  
 463. Typhlitis.  
 464. Dysentery.  
 465. Ulceration.  
 466. Perforation.  
 467. Abscess in the sub-peritoneal tissue.  
 468. Fecal abscess.  
 469. Fistula.  
     *a.* Fecal fistula. *Syn.*, Artificial anus.  
 (557.) Vesico-intestinal fistula.  
 470. Hemorrhage.  
 471. Melæna.  
 472. \*Dilatation.  
 473. \*Tyimpanites.  
 474. \*Obstruction.  
 475. Stricture.  
 476. Intussusception.  
 477. Internal strangulation.  
     *a.* Mesenteric.  
     *b.* Mesocolic.  
 478. Hernia.  
     *a.* Reducible.  
     *b.* Irreducible.  
     *c.* Obstructed.  
     *d.* Inflamed.  
     *e.* Strangulated.  
     1. Diaphragmatic.  
     2. Epigastric.  
     3. Ventral.  
     4. Umbilical.  
     5. Lumbar.  
     6. Inguinal.  
         *a.* Oblique.  
         *b.* Direct.  
         *c.* Incomplete.  
         *d.* Scrotal.  
         *e.* Congenital.  
     7. Femoral.  
     8. Obturator.  
     9. Perineal.  
     10. Pudendal.  
     11. Vaginal.  
     12. Ischiatic.  
 479. Diseases of hernial sacs.  
     *a.* Inflammation.  
     *b.* Fibrinous effusion with closure.  
     *c.* Suppuration.  
     *d.* Dropsy.  
     *e.* Movable bodies.  
     *f.* Laceration.  
 (491.) Cancer.  
 (501.) Colloid.  
 480. Non-malignant tumor.  
     *a.* Polypus.  
 481. Worms.  
 (1066.) Concretions.  
 (1066.) Foreign bodies.

- (1061-63.) Injuries.  
 482. Diarrhœa.  
 (17.) Simple cholera.  
 (18.) Malignant cholera.  
     *a.* Choleraic diarrhœa.  
 483. \*Paralysis.  
 484. Colic.  
 (902<sup>a</sup>.) Lead colic.  
 485. Constipation.

## DISEASES OF THE RECTUM AND ANUS.

486. Ulcer.  
 487. Abscess.  
 488. Fistula in ano.  
 (558.) Recto-vesical fistula.  
 (586.) Recto-urethral fistula.  
 (670.) Recto-vaginal fistula.  
 489. Hæmorrhoids.  
     *a.* Internal.  
     *b.* External.  
 490. Hemorrhage from rectum.  
 491. Fissure of the anus.  
 492. Prolapsus.  
 493. \*Stricture.  
 (49.) Cancer of the rectum.  
 (49.) Cancer of the anus.  
     Syphilis of rectum.  
     Condyloma of anus.  
 494. Non-malignant tumor of the rectum.  
     *a.* Polypus.  
 (1072, 73.) Injuries.  
 (1079.) Foreign bodies.  
 495. Neuralgia.  
 496. Spasm of the sphincter ani.  
 497. Pruritus ani.

## DISEASES OF THE LIVER.

498. Hepatitis.  
 499. Abscess.†  
 (28.) Pyæmic inflammation and abscess.  
 500. Acute atrophy.  
 501. Simple enlargement. *Syn.*, Congestion of the liver.  
 502. Thickening of the capsule.  
 503. Cirrhosis.  
 504. Fatty liver.  
 506. Fibroid deposit.  
 505. Lardaceous liver. *Syn.*, Amyloid disease of the liver. Waxy liver.‡  
 (48.) Syphilitic deposit.  
 (49.) Cancer.  
 (50.) Colloid.  
 507. Non-malignant tumor.  
 508. Cyst.  
 (531.) Tubercle.  
 509. Hydatid.  
 (1061-63.) Injuries.  
 510. Jaundice. *Syn.*, Icterus.  
 511. Obstruction of vena portæ.

\* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When abscess of the liver is associated with dysentery, injury, or any other condition, the fact should be stated.

‡ Such cases have been described under the name of Scrofulous disease of the liver.

**DISEASES OF THE HEPATIC DUCTS AND GALL-BLADDER.**

512. Inflammation.  
 513. Ulcer.  
 514. Perforation.  
     *a.* Biliary fistula.  
 515. Obstruction.  
 (49.) *Cancer*.  
 516. Gallstones.  
     *a.* Passage of gallstones through duct.  
 (1061-63.) *Injuries*.

**DISEASES OF THE PANCREAS.**

517. Abscess.  
 518. Obstruction of the duct.  
 (49.) *Cancer*.  
 (50.) *Colloid*.  
 519. Calculi.

**DISEASES OF THE SPLEEN.**

520. Splenitis.  
 521. Abscess.  
 (28.) *Pyæmic inflammation and abscess*.  
 522. Congestion. *Syn.*, Ague cake.  
 523. Fibrinous deposit.  
 524. Hypertrophy.  
     *a.* Leucocythæmia.

525. Lardaceous spleen. *Syn.*, Amyloid disease. Waxy spleen.  
 (49.) *Cancer*.  
 (50.) *Colloid*.  
 (53.) *Tubercle*.  
 526. Hydatid.  
 (1058.) *Rupture*.

**DISEASES OF THE PERITONEUM.**

527. Peritonitis.  
 (713.) *a.* *Puerperal peritonitis*.  
     *b.* Chronic peritonitis.  
     *c.* Suppurative peritonitis.  
 (53.) *d.* *Tubercular peritonitis*.  
     *e.* Adhesions of peritoneum.  
 528. \*Ascites.  
 (49.) *Cancer*.  
 (50.) *Colloid*.  
 529. Hydatid.  
 (1059-62.) *Injuries*.

**DISEASES OF THE MESENTERIC GLANDS.**

530. Inflammation.  
 531. Abscess.  
 532. Enlargement.  
 (49.) *Cancer*.  
 533. Non-malignant growth.  
 (53.) *Tubercle*.  
 (58.) *Tabes mesenterica*.

**DISEASES OF THE URINARY SYSTEM.****DISEASES OF THE KIDNEY.**

534. Bright's disease. *Syn.*, Albuminuria.  
     1. Acute Bright's disease. *Syn.*, Acute albuminuria. Acute desquamative nephritis. Acute renal dropsy.  
     2. Chronic Bright's disease. *Syn.*, Chronic albuminuria.  
     *Sub-divisions:*  
         *a.* Granular kidney. *Syn.*, Contracted granular kidney. Chronic desquamative nephritis. Gouty kidney.  
         *b.* Fatty kidney.  
         *c.* Lardaceous kidney. *Syn.*, Amyloid disease. Waxy disease.  
 535. Suppurative nephritis.  
 536. Abscess.  
 537. Pyelitis.  
 538. Fibrinous deposit.  
 539. Hydronephrosis.  
 540. Hypertrophy.  
 541. Atrophy.  
 (49.) *Cancer*.  
 542. Non-malignant tumor.  
 543. Simple cyst.  
 544. Urinary cyst (from injury).  
 (53.) *Tubercle*.  
 545. Entozoa

- a.* Hydatid.  
     *b.* *Eustrongylus gigas*.  
 546. Calculus.  
 547. Calculus in the ureter.  
 548. Malformations.  
 (1061-63.) *Injuries*.  
 549. \*Hæmaturia renalis.  
 550. \*Suppression of urine. *Syn.*, Ischuria renalis.  
 (47.) *Diabetes*. *Syn.*, Diabetes mellitus.  
 551. \*Diuresis.  
 552. Movable kidney.

**DISEASES OF THE BLADDER.**

553. Cystitis. *Syn.*, Catarrh of the bladder.  
     *a.* Acute.  
     \*i.b. Chronic.  
 554. Ulceration.  
 555. Suppuration.  
 556. Sloughing.  
 557. Vesico-intestinal fistula.  
 558. Recto-vesical fistula.  
 559. Vesico-vaginal fistula.  
 559. Hypertrophy.  
 560. \*Distension.  
     *a.* Sacculated bladder.  
     *b.* Rupture.  
 561. Inversion.  
 562. Extroversion.

\* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

563. Hernia.  
 (49.) *Cancer*.  
 564. Fibrous tumor.  
 565. Villous tumor.  
 566. Calculus.  
   *a.* Uric acid.  
   *b.* Urate of ammonia.  
   *c.* Uric oxide. *Syn.*, Xanthic oxide.  
   *d.* Oxalate of lime.  
   *e.* Cystic oxide.  
   *f.* Phosphate of lime.  
   *g.* Triple phosphate.  
   *h.* Fusible.  
   *i.* Carbonate of lime.  
   *k.* Fibrinous.  
   *l.* Urosteolith.  
   *m.* Blood calculus.  
   Foreign bodies.  
 567. \*Hæmaturia (vesical).  
 568. \*Paralysis.  
 569. \*Irritability.  
 570. \*Spasm.  
 571. Neuralgia.  
 572. \*Incontinence of urine.  
 573. \*Retention of urine.

## DISEASES OF THE PROSTATE GLAND.

574. Inflammation.  
   *a.* Acute.  
   *b.* Chronic.  
 575. Ulceration.  
 576. Abscess.  
 577. Atrophy.  
 (49.) *Cancer*.  
 578. Non-malignant tumor. *Syn.*, Enlarged lobe of prostate.  
 579. Cyst.  
 (71.) *Tubercle*.  
 580. Calculi.

## GONORRHOEA AND ITS COMPLICATIONS.

581. Gonorrhœa.  
   *a.* In male.  
   *b.* In female.  
 582. Balanitis.  
   *Herpes preputialis*.  
 583. Phimosi.  
 584. Paraphimosis.  
 585. Condyloma.  
   *a.* In male.  
   *b.* In female.  
 586. Gleet.  
 587. Lacunar abscess.  
 588. Bubo.  
   Prostatic abscess.  
 589. Epididymitis. *Syn.*, Gonorrhœal orchitis.  
   *a.* Abscess.  
 590. Abscess of the spermatic cord.  
 (628.) *Inflammation of ovary*.  
 591. Abscess of the vulva.  
 (117.) *Gonorrhœal ophthalmia*.  
 (140.) *Gonorrhœal iritis*.  
 (86.) *Gonorrhœal rheumatism*.

## DISEASES OF THE MALE URETHRA.

592. Stricture.†  
   *a.* Organic.  
   *b.* Traumatic.  
   *c.* Spasmodic.  
   *d.* Inflammatory.  
 593. Ulcer.  
 594. Urinary abscess.  
 595. Urinary fistula.  
 596. Recto-urethral fistula.  
 597. Extravasation of urine.  
 598. Impacted calculus.  
   *a.* Foreign bodies.

## DISEASES OF THE GENERATIVE SYSTEM.

## DISEASES OF THE MALE ORGANS OF GENERATION.

## PENIS.

599. Inflammation.  
 600. Abscess.  
 (581<sup>a</sup>.) *Gonorrhœa*.  
 601. Gangrene.  
 602. \*Priapism.  
 (48<sup>l</sup>.) *Syphilis*.  
 (49<sup>l</sup>.) *Cancer*.  
   *a.* Of prepuce.  
   *b.* Of body.  
 603. Non-malignant tumors.  
 (1069.) *Injuries*.  
 604. Phimosi—congenital.

## SCROTUM.

605. Sloughing.

606. Œdema.  
 607. Elephantiasis.  
 (828.) *Prurigo*.  
 (48<sup>l</sup>.) *Syphilis*.  
 (49<sup>l</sup>.) *Cancer*.  
 (49<sup>c</sup>.) *Epithelial cancer*. *Syn.*, Chimney-sweeper's cancer.  
 608. Non-malignant tumor.

## CORD.

609. Hydrocele.  
   *Varieties:*  
   *a.* Encysted.  
   *b.* Diffused.  
 610. Varicocele.

\* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When the cause of the stricture is known it should be stated.



611. Non-malignant tumor.  
612. Neuralgia.

## TUNICA VAGINALIS.

613. Inflammation.  
614. Hydrocele.  
    *Varieties:*  
        *a.* Congenital.  
        *b.* Infantile.  
        *c.* Encysted.  
615. Hæmatocele.  
616. Loose bodies.

## TESTICLE.

617. Orchitis.

- a.* Acute.  
        *b.* Chronic.  
618. Abscess.  
619. Protrusion of tubuli. *Syn.*, Hernia testis. Fungus testis.  
620. Atrophy.  
    (48<sup>1</sup>.) *Syphilitic disease.*  
    (49<sup>1</sup>.) *Cancer.*  
621. Non-malignant tumor.  
622. Cystic disease.  
    (53<sup>2</sup>.) *Tubercle.*  
    (1069.) *Injuries.*  
623. Fœtal remains in testicle.  
624. Malposition.  
625. Spermatorrhœa.  
626. Impotence.  
627. Neuralgia.

## DISEASES OF THE FEMALE ORGANS OF GENERATION IN THE UNIMPREGNATED STATE.

## OVARY.

628. Inflammation.  
629. Abscess.  
630. Hemorrhage.  
631. Atrophy.  
632. Hypertrophy.  
    (49.) *Cancer.*  
633. Fibrous tumor.  
634. Encysted dropsy.  
635. Complex cystic tumors. *Syn.*, Alveolar, gelatinous, and colloid tumor of the ovary. Cysto-sarcoma.  
    *a.* With intracystic growths.  
636. Cysts, containing tegumentary structures—hair, teeth, and bones.  
    *Syn.*, Dermoid cysts.  
    (58<sup>1</sup>.) *Tubercle.*  
637. Cyst containing hydatid.  
638. Dislocation.  
639. Hernia.

## FALLOPIAN TUBE.

640. Abscess.  
641. Dropsy.  
642. Stricture.  
643. Occlusion.  
    (49<sup>1</sup>.) *Cancer.*  
644. Cyst.  
    (53<sup>1</sup>.) *Tubercle.*  
645. Dislocation.  
646. Hernia.

## BROAD LIGAMENT.

647. Inflammation.  
    *a.* Pelvic peritonitis.  
    *b.* Pelvic cellulitis.  
648. Abscess.  
649. Cyst.

- (817.) Peri-uterine or pelvic hæmatocele.

## UTERUS.

650. Catarrh. *Syn.*, Leucorrhœa.  
    *a.* Hydrorrhœa.  
651. Inflammation.  
    Granular inflammation.  
    Abrasion.  
652. Ulcer.  
653. Rodent ulcer.  
654. Abscess.  
655. Utero-vesical fistula.  
656. Stricture of the orifice.  
657. " of the canal.  
658. Occlusion of the orifice.  
659. " of the canal.  
660. Hypertrophy.  
661. Atrophy.  
    (49<sup>1</sup>.) *Cancer.*  
        *a.* *Scirrhus.*  
        *b.* *Medullary cancer.*  
        *c.* *Epithelial cancer.*  
662. Fibrous tumor.  
663. Polypus.\*  
    (53<sup>1</sup>.) *Tubercle.*  
664. Displacements and distortions.  
    *a.* Antiversion.  
    *b.* Retroversion.  
    *c.* Antiflexion.  
    *d.* Retroflexion.  
    *e.* Inversion.  
    *f.* Prolapsus.  
        1. Procidentia.  
    *g.* Hernia.

## VAGINA.

665. Catarrh. *Syn.*, Leucorrhœa.  
666. Inflammation.

\* Under this head should be returned all pedunculated tumors growing from the cavity or neck of the uterus, whether mucous, cellular, or fibrous.



667. Abscess.  
 (580<sup>b</sup>.) *Gonorrhœa*.  
 668. Cicatrix or band.  
 668<sup>a</sup>. Vaginal fistula.  
 669. Vesico-vaginal fistula.  
 670. Recto-vaginal fistula.  
 671. Hernia.

a. Cystocele.

b. Rectocele.

(49<sup>1</sup>.) *Cancer*.

672. Laceration.

673. Polypus.

#### VULVA.

- 673<sup>a</sup>. Inflammation of labia.  
 674. Pruritus of labia.  
 (853.) *Eczema of labia*.  
 675. Œdema of labia.  
 676. Abscess.  
 677. Gangrene.  
 678. Hypertrophy.\*  
 a. Elongation of cervix.  
 679. Occlusion.  
 680. Imperforate hymen.  
 (262.) *Varicose veins*.  
 (48<sup>1</sup>.) *Syphilis*.

(49<sup>1</sup>.) *Cancer*.

681. Vascular tumor of the meatus urinarius.

682. Mucous cyst.

(585<sup>b</sup>.) *Condyloma*.

#### FUNCTIONAL DISEASES.

683. Amenorrhœa. *English Syn.*, Absent menstruation.

*Varieties:*

a. From original defective formation.

b. From want of development at the time of puberty.

c. From mechanical obstruction.

d. From temporary suppression.

688. *Chlorosis*. *English Syn.*, *Green sickness*.

684. Scanty menstruation. *Syn.*, Deficient menstruation.

685. Vicarious menstruation.

686. Dysmenorrhœa. *English Syn.*, Painful menstruation.

687. Menorrhagia. *English Syn.*, Excessive menstruation.

#### AFFECTIONS CONNECTED WITH PREGNANCY.

##### †DISORDERS OF THE NERVOUS SYSTEM.

Neuralgia.

*Varieties:*

a. Odontalgia.

b. Cephalalgia.

c. Mastodynia.

Chorea.

Convulsions.

Hypochondriasis.

Mania.

##### †DISORDERS OF THE CIRCULATORY SYSTEM.

Varicose veins—

a. Of the lower extremities.

b. Of the labia.

c. Of the rectum. *Hæmorrhoids*.

Serous exudation.

*Varieties:*

a. Ascites.

b. Œdema of labia.

c. Œdema of lower extremities.

Syncope.

Palpitation.

##### †DISORDERS OF THE RESPIRATORY SYSTEM.

Dyspnœa.

Orthopnœa.

Cough.

##### †DISORDERS OF THE DIGESTIVE SYSTEM.

Salivation.

Depraved and capricious appetite.

Nausea and vomiting.

Cardialgia or Heartburn.

Pyrosis.

Intestinal cramp—colic.

Constipation.

Diarrhœa.

Jaundice.

##### †DISORDERS OF THE URINARY SYSTEM.

Albuminuria.

Dysuria.

Incontinence of urine.

Retention of urine.

#### DISORDERS OF THE GENERATIVE SYSTEM.

689. Metritis. *Syn.*, Hysteritis.

690. Discharge of watery fluid from the uterus. *Hydorrhœa*.

691. Rheumatism of the uterus.

692. Hysteralgia.

693. Spurious pains and cramps.

(665.) *Catarrh of vagina*. *Syn.*, *Leucorrhœa*.

694. Sanguineous discharge. *Syn.*, Menstruation.

695. Hemorrhage.

696. Displacement of uterus.

*Varieties:*

a. Prolapsus.

b. Hernia.

c. Retroversion.

(674.) *Pruritus of the vulva*.

697. Abortion.

\* Specify the part.

† These affections are secondary, and are therefore not numbered.

698. Premature labor.  
699. Extra-uterine gestation.

#### AFFECTIONS CONNECTED WITH PARTURITION.

700. Atony of the uterus.  
701. Over-distension of the uterus.  
    *a.* From excess of liquor amnii.  
    *b.* " twins, triplets, &c.  
702. Mechanical obstacle to the action of the uterus.  
    *a.* From occlusion of the os uteri.  
    *b.* From rigidity of the—  
        os uteri.  
        vagina.  
        perineum.  
    *c.* From cancer of the cervix uteri.  
    *d.* " narrowness of the vagina.  
    *e.* " cicatrix or band in the vagina.  
    *f.* From vaginal cyst.  
    *g.* " prolapsus of the bladder.  
    *h.* " stone in the bladder.  
    *i.* " distended rectum.  
    *k.* " prolapsus of the rectum.  
    *l.* " tumor.  
    *Varieties:*  
        1. Uterine.  
        2. Ovarian.  
        3. Pelvic.  
        4. Of external parts.  
    *m.* From polypus.  
    *n.* " fractured pelvis.  
    *o.* " exostosis.  
    *p.* From distorted or contracted pelvis.  
    *q.* From dislocated lumbar vertebræ into pelvis. *Syn.*, Spondylo listhesis.  
    *r.* From ankylosed coccyx.  
    *s.* From diminutive pelvis.  
    *t.* Extreme anteversion of uterus (with pendulous abdomen).  
    *u.* From excessive size of fœtus.  
    *v.* " malposition of fœtus.  
    *w.* " malformation of fœtus.  
    *x.* From enlargement of fœtus from disease.  
    *y.* From unusual thickness of foetal membranes.  
    *z.* From unusual shortness of funis.  
703. Hemorrhage.  
    *a.* From placenta prævia. *Syn.*, Unavoidable hemorrhage.  
    *b.* From accidental detachment of

- placenta. *Syn.*, Accidental hemorrhage.  
    *c.* Thrombus of cervix uteri or labium.  
704. Rupture or laceration of the—  
    uterus.  
705. vagina.  
706. urinary bladder.  
707. perineum.  
708. Retention of the placenta.  
    *a.* From atony of the uterus.  
    *b.* From irregular or hour-glass contraction.  
    *c.* From preternatural adhesions.  
709. Inversion of the uterus.  
710. Convulsions.

#### AFFECTIONS CONSEQUENT ON PARTURITION.

711. Post-partum hemorrhage.  
(83.) *Puerperal ephemera.*  
712. Milk fever.  
(82.) *Puerperal fever.*  
713. Metro-peritonitis. *Syn.*, Puerperal peritonitis.  
    *a.* Metritis.  
    *b.* Peritonitis.  
(257.) *Phlebitis.*  
(258.) *Phlegmasia dolens.*  
714. Pelvic cellulitis.  
715. Iliac and pelvic abscesses.  
716. Sloughing of cervix uteri.  
717. " vagina.  
718. " perineum.  
719. " bladder.  
720. " rectum.  
(655.) *Utero-vesical fistula.*  
(669.) *Vesico-vaginal fistula.*  
(670.) *Recto-vaginal fistula.*  
(723.) *Mammary inflammation.*  
(724.) *Mammary abscess.*  
721. Puerperal mania.  
    *a.* Connected with parturition.  
    *b.* " " lactation.  
722. Puerperal convulsions. *Syn.*, Eclampsia.  
723. Sudden death after delivery.  
    *a.* From shock or nervous exhaustion.  
    *b.* From impaction of coagula in the heart and great vessels.  
        1. Thrombosis.  
        2. Embolism.  
    *c.* From entrance of air into veins (from separation of placenta)  
(896.) *Still-born.*  
(897.) *Premature birth.*

#### DISEASES OF THE FEMALE BREAST.

- 728<sup>a</sup>. Inflammation.  
    *a.* Acute.  
    *b.* Chronic.  
724. Abscess.  
725. Sinus.  
726. Galactorrhœa.  
727. Deficiency of milk.

728. Hypertrophy.  
729. Atrophy.  
730. Depressed nipple.  
731. Chapped nipple.  
732. Ulcerated nipple.  
(49<sup>1</sup>.) *Cancer.*  
    *a.* *Scirrhus.*

- b. *Medullary cancer.*
- c. *Epithelial cancer.*
- (50.) *Colloid.*
- 733. Fibrous tumor. *Syn.*, Painful subcutaneous tumor.
- 734. Fibro-plastic tumor.
- 735. Fatty tumor.
- 736. Osseous tumor.
- 737. Cartilaginous tumor. *Syn.*, Enchondroma.

- 738. Chronic mammary tumor. *Syn.*, Adenoid tumor.
- 739. Vascular tumor.
- 740. Cyst.
- 741. Complex cystic tumor. *Syn.*, Cystosarcoma.
- 742. Hydatid.
- 743. Hyperæsthesia.
- 744. Mastodynia. *Syn.*, Neuralgia.

## DISEASES OF THE MALE MAMMILLA.

- 745. Inflammation.
- 746. Hypertrophy.
- (49<sup>1</sup>.) *Cancer.*

- 747. Non-malignant tumor.
- 748. Cyst.

## DISEASES OF THE ORGANS OF LOCOMOTION.

## DISEASES OF BONES.\*

- 749. Ostitis.
  - a. Periostitis.
    - 1. Nodes.
- 750. Diffuse periostitis. *Syn.*, Acute periosteal abscess.
  - a. Acute necrosis.
- 751. Osteo-myelitis.
- 752. Chronic abscess.
- 753. Caries.
- 754. Necrosis.
- 755. Mollities ossium.
- 756. Hypertrophy.
- 757. Atrophy.
- 758. Spontaneous fracture. (The cause, if known, should be stated.)
  - (48<sup>1</sup>.) *Syphilitic disease.*
  - (49<sup>1</sup>.) *Cancer.*
- 759. Non-malignant tumors.
  - a. Fibrous and fibro-cystic.
  - b. Myeloid.
  - c. Cartilaginous. *Syn.*, Enchondroma.
  - d. Exostosis.
    - 1. Diffused exostosis.
- 760. Cyst.
  - (54.) *Rickets.*
  - (53.) *Scrofulous disease.*
- 761. Hydatid.

## DISEASES OF JOINTS.†

- 762. Acute synovitis.
- 763. Chronic synovitis.
  - a. Pulpy degeneration of synovial membrane.
  - b. *Strumous disease of joints.*
    - 1. *Morbus coxæ.*
- 764. Ulceration of cartilage.
- 765. Abscess.
  - a. *Pyæmic abscess.*
- 766. Ankylosis.
  - a. Deformity from ankylosis.

- 767. Dropsy of joint.
  - (36.) *Gonorrhœal rheumatism.*
  - (37.) *Synovial rheumatism.*
  - (42.) *Gouty synovitis.*
  - (43.) *Chronic osteo-arthritis.* *Syn.*, *Chronic rheumatic arthritis.*
- 768. Degeneration of cartilage and articular surfaces of bones.
- 769. Perforation of joints.‡
- 770. Loose cartilage. *Syn.*, Loose body.
- 771. Relaxation of ligaments.
- 772. Displacement of articular cartilage.
- 773. Knock-knee.
- 774. Bow-leg, or out-knee.
- (49<sup>1</sup>.) *Cancer.*
- 775. Non-malignant tumor.
- 776. Neuralgia of joints.

## DISEASES OF THE SPINE.

- 777. Ulceration of ligaments and cartilages.
- 778. Caries and necrosis.
  - a. Spontaneous fracture of odontoid process.
- 779. Psoas, lumbar, and other abscesses.
- 780. Angular deformity. *Syn.*, Kyphosis.
- 781. Lateral curvature. *Syn.*, Skoliosis.
- 782. Anterior curvature. *Syn.*, Lordosis.
  - (54.) *Rickety curvature.*
- 783. Ankylosis.
  - (43.) *Chronic osteo-arthritis.*
- 784. Non-malignant tumor.
  - (49<sup>1</sup>.) *Cancer.*
- 785. Hydatid.
- 786. Deformity from malformations.

DISEASES OF THE MUSCULAR SYSTEM.§  
MUSCLE.

- 787. Inflammation.
- 788. Abscess.
- 789. Gangrene.
- 790. Atrophy.

\* In all cases the bone affected must be specified.

† In all cases the joint affected is to be specified.

‡ This refers to perforation by disease, and should be returned with the original affection.

§ In all cases the affected muscle or muscles should be stated.

791. Progressive atrophy.  
*Locomotor ataxy.*  
 792. Fatty degeneration.  
 793. Ossification.  
 (48<sup>1</sup>.) *Syphilitic deposit.*  
 (49<sup>1</sup>.) *Cancer.*  
 (50<sup>1</sup>.) *Colloid.*  
 794. Non-malignant tumor.  
*a. Erectile tumor.*  
 795. Cyst.  
*Rupture.*  
 (86.) *Infantile paralysis.*  
 796. Trichinosis.  
 (93.) *Spasm.*  
 797. Exhaustion.  
 (87<sup>b</sup>.) *Scrivener's palsy.*  
 (19<sup>a</sup>.) *Diphtheritic paralysis.*

## TENDONS.

798. Inflammation.  
 (875<sup>a</sup>.) *Thecal abscess.*  
 799. Adhesion of tendons.  
 (49<sup>1</sup>.) *Cancer.*  
 800. Non-malignant tumors.

801. Contraction of tendons, fasciæ, or muscles.  
 802. Club-foot.  
*a. Talipes varus.*  
*b. " valgus.*  
*c. " equinus.*  
*d. " calcaneus.*  
*e. " calcaneo-varus.*  
*f. " equino-valgus.*  
*Syn., Flat-foot.*  
 803. Club-hand.  
 804. Contracted palmar fascia.  
 805. Wry neck.  
 (1135.) *Rupture of tendon.*

## APPENDAGES OF MUSCULAR SYSTEM.

806. Enlarged bursa patellæ. *Syn., Housemaid's knee.*  
 807. Enlargement of other bursa (specify which).  
 808. Bursal tumor.  
 809. Bursal abscess.  
 810. Bunion.  
 811. Ganglion.  
*a. Diffused palmar ganglion.*

## DISEASES OF THE CELLULAR TISSUE.

812. Inflammation.  
 813. Abscess.  
 844. Inflammatory induration in the newly born.  
 815. Slough.  
 (27<sup>b</sup>.) *Phlegmonous erysipelas.*  
 (856.) *Carbuncle. Syn., Anthrax.*  
 816. Obesity.

817. Hemorrhage.  
*a. Pelvic hæmatocele.*  
 818. Non-malignant tumor.  
 (49<sup>1</sup>.) *Cancer.*  
 819. Parasites.  
 (135<sup>1</sup>.) *Foreign substances.*  
 820. Emphysema.

## DISEASES OF THE CUTANEOUS SYSTEM.

## DISEASES OF THE SKIN.\*

- (27.) *Erysipelas.*  
 821. Erythema. This term includes  
 1. Erythema læve.  
 2. Erythema fugax. *Syn., E. volaticum.*  
 3. Erythema marginatum.  
 4. " papulatum.  
 5. " tuberculatum.  
 6. " nodosum.  
 822. Intertrigo.  
 823. Roseola. (This term includes  
 1. Roseola æstiva.  
 2. " autumnalis.  
 3. " symptomatica.  
 4. " annulata.)  
 824. Urticaria. *English Syn., Nettle rash.*  
*a. Urticaria acuta.*  
*b. " chronica.*  
 (Under one or other of these heads are included  
 1. Urticaria febrilis.  
 2. " evanida.  
 3. " perstans.

4. Urticaria conferta.  
 5. " subcutanea.  
 6. " tuberculata.)  
 825. Pellagra.  
 826. Acrodynia.  
 827. Asturian rose.  
 828. Prurigo.  
 829. Lichen. (This term includes  
 1. Lichen simplex.  
 2. " pilaris.  
 3. " circumscriptus.  
 4. " agrius.  
 5. " tropicus. *English Syn., Prickly heat.*)  
 (The so-called Lichen lividus is really a form of Purpura.)  
 830. Strophulus. *English Syn., Red gum.*  
 Tooth rash. (This term includes  
 1. Strophulus intertinctus.  
 2. " confertus.  
 3. " candidus.)  
 (Strophulus albidus is referred to Acne.  
 " violaticus to Erythema.)

\* Where the disease is local its situation should be specified.

831. Pityriasis. (This term includes Pityriasis capitis. *English Syn.*, Dandriff.)  
(Pityriasis versicolor is referred to Parasitic affections as a *Synonym* of Tinea versicolor.)
832. Psoriasis. (This term includes Lepra.)
833. Psoriasis vulgaris. *Syn.*, Lepra vulgaris.  
a. Psoriasis guttata.  
b. " diffusa.  
c. " gyrata.  
d. " inveterata.
834. Miliaria.  
a. Sudamina.
835. Herpes.  
a. Herpes phlyctænodes.  
b. " circinatus.  
c. " iris.  
d. " zoster. *English Syn.*, Shingles.
836. Pemphigus. *Syn.*, Pompholyx.  
a. Pemphigus acutus.  
b. " chronicus.  
c. " solitarius.
837. Eczema.  
a. Eczema simplex.  
b. " rubrum.  
c. " impetiginodes.  
d. " chronicum.
838. Impetigo.  
a. Impetigo sparsa.  
b. " confluens.  
1. Figurata.  
2. Larvalis.
839. Rupia.  
a. Rupia simplex.  
b. " prominens.  
c. " escharotica. *Syn.*, Pemphigus gangrænosus.
840. Ecthyma.
841. Acne.  
a. Acne punctata.\*  
b. " indurata.  
c. " rosacea.  
d. " strophulosa. *Syn.*, Strophulus albidus.
842. Sycosis. *Syn.*, Mentagra.†
843. Stearrhœa.  
a. Stearrhœa simplex.  
b. " nigricans.
844. Ichthyosis.  
a. Ichthyosis vera.  
b. " cornea.
845. Xeroderma.
846. Leucoderma. (This term includes Vitiligo.)
847. Albinismus.
848. Canities.
849. Melasma.  
(282.) *Melasma Addisoni.* *English Syn.*, Addison's disease. *Bronze skin.*
850. Lentigo and Ephelis. *English Syn.*, Freckles.
851. Chilblain.
852. Frostbite.
853. Ulcer.
854. Fissures.  
(852.) *Cancrum oris.*
855. Boil.
856. Carbuncle. *Syn.*, Anthrax.  
(26.) *Malignant pustule.*
857. Onychia.
858. Onychia maligna.
859. Whitlow.  
a. Thecal abscess.
860. Gangrene.
861. Hypertrophy.
862. Corn.
863. Bunion.
864. Elephantiasis arabum. *Syn.*, Barbadoes leg; Elephas.  
(51.) True leprosy. *Syn.*, El. Græcorum.
865. Atrophy.  
a. Linear atrophy.  
b. Alopecia.  
c. Atrophy of nails.  
(49.) *Cancer.*
866. Fibro-cellular tumor.
867. Fatty tumor.  
(263.) *Nævus vascularis.*
868. Nævus. *Syn.*, Port wine stain.
869. Nævus pilaris. *Syn.*, Mole.
870. Sebaceous tumor.  
a. Steatoma.
871. Cornua.
872. Molluscum.
873. Warts.
874. Condyloma.  
a. Male.  
b. Female.
875. Cheloid.
876. Frambœsia.  
(51.) *Lupus.*  
(53.) *Scrofulous disease.*
877. Ingrown nail.
878. Silver-stain.  
(986.) *Burns and Scalds.*  
(101.) *Hyperæsthesia.*
879. Pruritus.  
(102.) *Anæsthesia.*
880. Ephidrosis.
881. Anidrosis.

## PARASITIC DISEASES OF THE SKIN.

882. Tinea tonsurans. *Syn.*, Ringworm.  
*Parasite*, Trichophyton tonsurans.
883. Tinea decalvans. *Syn.*, Alopecia areata. *Par.*, Microsporon Audouini.
884. Tinea favosa. *Syn.*, Favus. *Par.*, Achorion Schœnleinii, Pucciani Favi.
885. Tinea versicolor. *Syn.*, Pityriasis versicolor. *Par.*, Microsporon furfur.

\* When the Demodex folliculorum is discovered, its presence should be stated.

† When the Microsporon mentagrophytes is discovered, its presence should be stated.

886. *Tinea Polonica*. *Syn.*, *Plica Polonica*. *Par.*, *Trichophyton sporuloides*.
887. *Mycetoma*. *Syn.*, *Madura foot*. *Par.*, *Chinonyphe Carteri*.
888. *Scabies*. *Syn.*, *Itch*. *Par.*, *Sarcoptes scabiei*.
889. *Phthiriasis*, from *Phthirius inguinalis*. *English Syn.*, *Crab-louse*.
890. Irritation caused by  
*a. Pediculus capitis.*  
*b. " palpebrarum.*  
*c. " vestimenti.*  
*d. " tabescentium.*
891. *Pulex penetrans*. *English Syn.*, *Chigoe*.
- Pulex irritans.*
892. *Cimex*.
893. *Leptothrix autumnalis*. *English Syn.*, *Harvest-bug*.
894. Wasps, bees, and other stinging insects.
895. Nettles and other stinging plants.
- CONDITIONS NOT NECESSARILY ASSOCIATED WITH GENERAL OR LOCAL DISEASE.
896. Still-born.
897. Premature birth.
898. *Old age*.\*
899. †*Debility*.

## POISONS.

In returning cases of poisoning, the precise agent should be stated.

## METALS AND THEIR SALTS.

900. Arsenic.
901. Mercury.  
 (96.) *Mercurial tremor*.
902. Lead.  
*a. Lead colic. Syn., Painters' colic.*  
*b. Lead palsy.*  
*c. Blue gum.*  
*d. Metallic stain of conjunctiva (from lead).*
903. Copper.
904. Antimony.
905. Zinc.
906. Silver.  
*a. Silver-stain.*
907. Iron.
908. Bismuth.
909. Chromium.  
*a. (Bichromate of potash.)*

## CAUSTIC ALKALIES.

910. Potash.
911. Soda.
912. Ammonia.
913. Alkaline salts.

## METALLOIDS.

914. Phosphorus.
915. Iodine.

## ACIDS.

916. Sulphuric acid.
917. Nitric acid.
918. Muriatic acid.
919. Phosphorous acid.
920. Oxalic acid.
921. Tartaric acid.

## VEGETABLE POISONS.

922. Savin.

923. Croton oil.
924. Elaterium.
925. Colchicum.
926. Black hellebore.
927. White hellebore.  
*a. Veratria.*
928. Squill.
929. Ergot of rye.  
 (46.) *a. Ergotism.*
930. Opium.
931. Indian hemp. (*Cannabis indica*.)
932. Alcohol.  
 (103.) *a. Delirium tremens.*
933. Ether vapor.
934. Chloroform vapor.
935. Henbane. (*Hyoscyamus*.)
936. Deadly nightshade. (*Belladonna*.)  
*a. Atropia.*
937. Thorn apple. (*Stramonium*.)
938. Prussic acid.  
*a. Oil of bitter almonds.*  
*b. Laurel water.*
939. Cyanide of potassium.
940. Nitro-benzole.
941. Wourali Curara. (*Strychnos toxifera*.)
942. Hemlock. *Conium*.
943. Monkshood. (*Aconium*.)  
*a. Aconita.*
944. Foxglove. (*Digitalis*.)  
*a. Digitalin.*
945. Tobacco.  
*a. Nicotin.*
950. Hemlock dropwort. (*Oenanthe crocata*.)
946. Nux vomica.  
*a. Strychnia.*  
*b. Brucia.*
947. Upas tieute. (*Strychnos tieute*.)
948. Upas antiar.

\* This mode of return is only to be employed when the cause of death is not traceable to definite disease.

† When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

949. Calabar bean. (*Physostigma venenosum*.)  
 951. Fool's parsley. (*Æthusa cynapium*.)  
 952. Water hemlock. (*Cicuta virosa*.)  
 953. Camphor.  
 954. Cocculus Indicus.  
 955. Darnel. (*Lolium temulentum*.)  
 956. Indian tobacco. (*Lobelia inflata*.)  
 957. Laburnum.  
 958. Yew. (*Taxus baccata*.)  
 959. Poisonous fungi.  
     *a.* Mouldy bread.

## ANIMAL POISONS.

960. Spanish fly. (*Cantharides*.)  
 961. Decayed and diseased meat.  
 962. Poisonous meat.  
     *a.* Sausages.  
 963. Poisonous cheese.  
 964. Poisonous milk.  
 965. Poisonous fish.  
     *a.* Mussels.

## GASEOUS POISONS.

966. Ammonia.  
 967. Nitrous acid vapor.  
 968. Chlorine.  
 969. Carbonic acid.  
 970. Carbonic oxide.  
 971. Coal gas.

972. Cyanogen.  
 973. Sulphuretted hydrogen.  
 974. Putrid and morbid exhalations.  
 975. Other noxious effluvia.

## MECHANICAL IRRITANTS.

976. Pounded glass.  
 977. Steel filings.

## POISONED WOUNDS.

## Varieties:

979. *a.* By venomous animals.  
     1. Snakes.  
     2. Scorpions.  
     3. *Stinging insects*.  
 (894.) *b.* By animals having infectious disease.  
     (23.) *Glanders*.  
     (24.) *Farcy*.  
     (25.) *Equina mitis*.  
     (26.) *Malignant pustule*.  
     (89.) *Hydrophobia*.  
     (2.) *Cow-pox*.  
 980. *c.* By dead animal matter.  
 981. *d.* By morbid secretions.  
 982. *e.* By vegetable substances.  
 983. 1. Poisoned arrows.  
     *Wourali*.  
 984. 2. Subcutaneous injection.\*  
 985. *f.* By mineral substances.

## INJURIES.

## GENERAL INJURIES.

986. Burns and scalds (including explosions).†  
 987. Lightning stroke.  
 988. Multiple injury. (The cause and extent to be stated.)  
 989. Asphyxia.  
     *a.* Drowning.

- b.* Hanging.  
*c.* Strangling.  
*d.* Overlying.  
*e.* *Gaseous Poisons*.  
 990. Privation.‡  
 991. Exposure to cold.‡  
 992. Infant exposure.‡  
 993. Neglect.‡

## LOCAL INJURIES.§

## INJURIES OF THE HEAD AND FACE.

## A.—OF THE HEAD.

994. Contusion.  
     *a.* Cephalhæmatoma.  
 995. Scalp-wound: bone not exposed.  
 996. " bone exposed.  
 997. Concussion of the brain.  
 998. Fracture of the vault of the skull.  
     *a.* Simple, without depression.  
     *b.* " with depression.

- c.* Compound, without depression.||  
*d.* " with depression.  
 999. Hernia cerebri.  
 1000. Fracture of the base of the skull.  
 1001. Wound of the skull.¶  
 1002. Laceration of the brain, without fracture.  
 1003. Injuries of the vessels. (Specify which.)  
 1004. Injuries of the cerebral nerves.

\* In returning such cases, specify the agent employed.

† When limited to one part of the body, the part is to be specified: *e. g.*, Scald of the larynx.

‡ Any affection that may have been induced by this cause ought to be stated.

§ In all cases of injury, specify whether accidental, judicial, homicidal, self-inflicted, or in battle.

|| In such cases state the main features of the case in the fewest words possible.

¶ If from gunshot, to be stated.



**B.—OF THE FACE.**

- 1005. Contusion.
- 1006. Wound.\*
- 1007. Foreign bodies in the ear.
- 1008.   "       "       nose.
- 1009.   "       "       antrum.
- 1010.   "       "       soft parts.
- 1011. Fracture of the facial bones.
- 1012.   "       lower jaw.†
- 1018. Dislocation of the jaw.

**INJURIES OF THE EYE.**

- 1014. Contusions.
- 1015. Contusions, with rupture of the sclerotic. *Syn.*, ruptured globe.
- 1016. Contusion, with dislocation of the lens.
- 1017. Contusion, with hemorrhage into the globe.
- 1018. Foreign bodies in the cornea or conjunctiva.
- 1019. Foreign bodies in the cavity of the eye.‡
- 1020. Wounds of the eyelids.
- 1021.   "       conjunctiva.
- 1022.   "       sclerotic.
- 1023.   "       cornea.
- 1024.   "       lens.
- 1025.   "       iris.
- 1026. Dislocation of the globe.
- (158.) *Total disorganization of the eye from injury.*
- 1027. Wounds and injuries of the parts within the orbit.
- 1028. Chemical injuries of the eyelids and eye.
- 1029. Burns and scalds.

**INJURIES OF THE NECK.**

- 1030. Contusion of the soft parts.
- 1031. Fracture of the hyoid bone.
- 1032.   "       cartilages of the larynx.
- 1033. Rupture of the trachea.
- 1034. Dislocation of the hyoid bone.
- 1035. Wound.
  - a. Superficial.
  - b. Cut throat.\*
  - c. Gun-shot.\*
  - d. Of great vessels.\*
  - e. From the mouth.
- (986.) *Burn and scald of larynx.*
- 1036. Foreign bodies in the air-passages.
- 1037.   "       "       pharynx.
- 1038.   "       "       œsophagus.
- 1039. Injury of pharynx and œsophagus by corrosive substances.

**INJURIES OF THE CHEST.\***

- 1040. Contusion.
- 1041. Fracture of the ribs (including costal cartilages) without injury to lung.
- 1042. Fracture of the ribs (including costal cartilages) with injury to lung.
- 1043. Fracture of sternum.
- 1044. Wound of parietes.
- 1045. Perforating wound of chest.\*‡
- 1046. Perforating wound of pleura or lung.\*‡
- 1047. Wound of anterior mediastinum.\*‡
- 1048. Wound of pericardium and heart.\*‡
- 1049. Wound of vessels.\*‡
- 1050. Rupture of heart or lung without wound or fracture.\*

**INJURIES OF THE BACK. (Including the whole spinal region.)**

- 1051. Contusion.
- 1052. Sprain.
- 1053. Wound.\*‡
- 1054. Fracture and dislocation of spine.||
- 1055. Injury of the cord without known fracture.

**INJURIES OF THE ABDOMEN.**

- 1056. Contusion.
- 1057. Contusion with rupture of muscles.\*
- 1058. Contusion with rupture of viscera.
- 1059. Wound of parietes.‡
- 1060. Wound of parietes, with protrusion of uninjured viscera.
- 1061. Wound of parietes, with protrusion of wounded viscera.
- 1062. Wound of parietes with wound of unprotruded viscera.
- 1063. Wound of viscera without wound of parietes.\*
- 1064. Foreign bodies in the peritoneal cavity.
- 1065. Foreign bodies in the stomach.
- 1066. Foreign bodies and concretions in the intestines.
- 1067. Fistula from injury, and artificial anus.

**INJURIES OF THE PELVIS.**

- 1068. Contusion.
- 1069. Wound of the male perineum, scrotum, and penis.‡
- 1070. Wound of the female perineum and vulva.
- 1071. Wound of the vagina and internal female organs.\*

\* In such cases, state the main features of the case in the fewest words possible.

† Injuries of the alveoli and teeth are to be returned with the other affections of those parts.

‡ If from gunshot, to be so stated.

§ Specify when from gunshot.

|| The seat of the injury, and the existence and extent of paralysis, to be stated.



1072. Wound of the rectum.\*  
 1078. " anus.  
 1074. " bladder.  
 1075. Rupture of bladder without wound.  
 1076. Rupture of bladder from fracture.†  
 1077. Injuries of the pregnant uterus.  
 1078. Foreign bodies in vagina.  
 1079. " " rectum.  
 (566, 598.) " " *bladder and urethra.*‡  
 1080. Fracture and dislocation of pelvis.  
 1081. Fracture and dislocation of pelvis, with rupture of bladder or urethra.

#### INJURIES OF THE UPPER EXTREMITIES.

1082. Contusion.  
 1083. Wound.\*§  
 1084. Wound of joint.  
 1085. " vessels.\*  
 1086. Sprain. (Specify which joint.)  
 1087. Dislocation. (When compound, to be so stated.)  
 1088. Dislocation of sterno-clavicular joint.  
 1089. Dislocation of acromio-clavicular joint.  
 1090. Dislocation of shoulder.  
 1091. Dislocation of elbow.  
 1092. " wrist and carpus.  
 1093. " thumb.  
 1094. " phalangeal joints.  
 1095. Separation of epiphyses.  
 1096. Foreign bodies embedded.\*  
 1097. Greenstick fracture, or bending of bone. (Specify which bone.)  
 1098. Fracture. (State whether simple or compound.)  
 1099. " of clavicle.  
 1100. " scapula.  
 1101. " humerus.  
 1102. " forearm.  
 1103. " carpus, metacarpus, and phalanges.  
 1104. Ununited fracture, or false joint. (Specify which bone.)

#### INJURIES OF THE LOWER EXTREMITIES.

1105. Contusion.  
 1106. Sprain. (Specify which joint.)  
 1107. Wound.\*§  
 1108. " of joint.  
 1109. " of vessels.\*  
 1110. Foreign bodies embedded.\*  
 1111. Separation of epiphyses.  
 Fracture. (When compound, to be so stated.)  
 1112. " of femur.  
 1113. " of cervix femoris.  
 1114. " Intra capsular.  
 1115. Fracture of trochanter major.  
 1116. " patella.  
 1117. " leg, both bones.  
 1118. " " tibia alone.  
 1119. " " fibula alone.  
 1120. " bones of the foot.  
 Dislocation. (When compound, to be so stated.)  
 1121. " of hip.  
 1122. " patella.  
 1123. " knee.  
 1124. " head of fibula.  
 1125. " foot, at the ankle.  
 1126. " " at calcaneo-astragaloid, and scapho-astragaloid joints.  
 1127. " astragalus.  
 1128. " os calcis.  
 1129. " other tarsal bones.  
 1130. " metatarsus, and phalanges.  
 1131. Ununited fracture, or false joint. (Specify which bone.)

#### INJURIES OF THE ABSORBENT SYSTEM.

1132. Foreign bodies and concretions.  
 1133. Wounds of lymphatics.  
 1134. Rupture of muscle.  
 1135. " tendon.  
 1136. Foreign substances in cellular tissue.

\* In such cases, state the main features of the case in the fewest words possible.

† Rupture of bladder from accumulation of urine is usually from stricture, and must be returned under the appropriate heading.

‡ Return such cases with calculus in the bladder and urethra.

§ Specify when from gunshot.

## HUMAN PARASITES.

The Parasites are to be returned under Local Diseases.

## SUBDIVISIONS.

1. ENTOTZOA.
2. ECTOTZOA.
3. ENTOPHYTA AND EPIPHYTA.

## ENTOTZOA.

## CLASSES.

- A. CŒLELMINTHA. *English Syn.*, Hollow worms.
- B. STERELMINTHA. *English Syn.*, Solid worms.
- C. ACCIDENTAL PARASITES.

## CLASS A.—CŒLELMINTHA.

- Ascaris lumbricoides*. (Linnæus.) *Habitat*, Intestines.  
*Ascaris mystax*. (Rudolphi.) *Hab.*, Intestines.  
*Trichocephalus dispar*. (Rudolphi.) *Hab.*, Intestines.  
*Trichina spiralis*. (Owen.) *Hab.*, Muscles.  
*Filaria Medinensis*. (Gmelin.) *English syn.*, Guinea-worm. *Hab.*, Skin and subcutaneous tissues.  
*Filaria oculi*. (Nordmann.) *Syn.*, *Filaria lentis*. (Diesing.) *Hab.*, Eye.  
*Strongylus bronchialis*. (Cobbold.) *Hab.*, Bronchial tubes.  
*Eustrongylus gigas*. (Diesing.) *Hab.*, Kidney; intestines.  
*Sclerostoma duodenale*. (Cobbold.) *Syn.*, *Anchylostomum duodenale*. *Hab.*, Duodenum.  
*Dactylius aculeatus*. (Curling.) *Hab.*, Bladder.  
*Oxyuris vermicularis*. (Bremser.) *English syn.*, Thread-worm. *Hab.*, Rectum.

## CLASS B.—STERELMINTHA.

- Bothriocephalus latus*. (Bremser.) *Hab.*, Intestines.  
*Bothriocephalus cordatus*. (Leuckart.) *Hab.*, Intestines.  
*Tænia solium*. (Linnæus.) *Hab.*, Intestines.  
*Cysticercus* of *Tænia solium*. *Syn.*, *Cysticercus telæ cellulose*.  
*Tænia mediocanellata*. (Küchenmeister.) *Hab.*, Intestines.

- Tænia acanthotrias*. (Weinland.) *Hab.*, Intestines.  
*Tænia flavopuncta*. (Weinland.) *Hab.*, Intestines.  
*Tænia nana*. (Siebold.) *Hab.*, Intestines.  
*Tænia lophosoma*. (Cobbold.) *Hab.*, Intestines.  
*Tænia elliptica*. (Batsch.) *Hab.*, Intestines.  
*Cysticercus* of the *Tænia marginata*. *Syn.*, *Cysticercus tenuicollis*.  
*Echinococcus hominis*, or Hydatid of the *Tænia echinococcus*. (Siebold.)  
*Fasciola hepatica*. (Linnæus.) *Hab.*, Liver.  
*Distoma crassum*. (Busk.) *Hab.*, Duodenum.  
*Distoma lanceolatum*. (Mehlis.) *Hab.*, Hepatic duct; bowels.  
*Distoma ophthalmobium*. (Diesing.) *Hab.*, Eye.  
*Distoma heterophyes*. (Siebold.) *Hab.*, Small Intestines.  
*Bilharzia hæmatobia*. (Cobbold.) *Hab.*, Portal and venous blood.  
*Tetrastoma renale*. (Della Chiaje.) *Hab.*, Tubes of kidney.  
*Hexathyridium venarum*. (Treutler.) *Hab.*, Venous blood.  
*Hexathyridium pinguiola*. (Treutler.) *Hab.*, Ovary.

## CLASS C.—ACCIDENTAL PARASITES.

- Pentastoma denticulatum*. (Siebold.) *Hab.*, Liver; small intestines.  
*Pentastoma constrictum*. *Hab.*, Liver.  
*Cestrus hominis*. (Say.) *English syn.*, Larva of the gad-fly. *Hab.*, Intestines.  
*Anthomyia canicularis*. (A. Farre.) *Hab.*, Intestines.

## ECTOTZOA.

- Phthirius inguinalis*. (Leach.) *English syn.*, Crab-louse.  
*Pediculus capitis*. (Nitzsch.)  
*Pediculus palpebrarum*. (Le Jeune in Guillemeau.)  
*Pediculus vestimenti*. (Nitzsch.) *English syn.*, Body-louse.  
*Pediculus tabescentium*. (Burmeister.)  
*Sarcoptes scabiei*. (Latreille.) *Syn.*, *Acarus*. *English syn.*, Itch-insect.\*

\* To be returned amongst the parasitic diseases of the skin.

*Demodex folliculorum.* (Owen.)  
*Pulex penetrans.* (Gmelin.) *Syn.*, Chigoe.

#### ENTOPHYTA AND EPIPHYTA.

*Leptothrix buccalis.* (Wedl. Robin.)  
*English syn.*, Alga of mouth.  
*Oidium albicans.* (Link.) *English syn.*,  
 Thrush fungus. *Hab.*, Mouth in cases  
 of thrush, and certain mucous and cu-  
 taneous surfaces.  
*Sarcina ventriculi.* (Goodsir.) *Hab.*,  
 Stomach.  
*Torula cerevisiæ.* (Turpin.) *Syn.*, *Cryp-*  
*tococcus cerevisiæ.* (Kützing.) *Eng-*  
*lish syn.*, Yeast plant. *Hab.*, Stomach,  
 bladder, &c.

*Chionyphe Carteri.* *Hab.*, Deep tissues,  
 and bones of the hands and feet.  
*Achorion Schönleini.* (Remak.) *Hab.*,  
*Tinea favosa.\**  
*Puccinia favi.* (Ardsten.) *Hab.*, *Tinea*  
*favosa.\**  
*Achorion Lebertii.* (Robin.) *Syn.*, *Tri-*  
*chophyton tonsurans.* (Malmsten.)  
*Hab.*, *Tinea tonsurans.\**  
*Microsporon Audouini.* (Gruby.) *Hab.*,  
*Tinea decalvans.\**  
*Trichophyton sporuloides.* (Von. Wal-  
 ther.) *Hab.*, *Tinea polonica.*  
*Microsporon furfur.* (Eichstädt.) *Hab.*,  
*Tinea versicolor.\**  
*Microsporon mentagrophytes.* (Gruby.)  
*Hab.*, Follicles of hair in Sycosis or  
*Mentagra.\**

The foregoing list might be extended by the addition of various parasitic vegetations, which have been reported under the names of Algæ, Fungi, Mycodermis, Leptomiti, &c., but the characters or the existence of which are still the subject of inquiry.

### CONGENITAL MALFORMATIONS OF THE FŒTUS.

#### MALFORMATIONS RESULTING FROM INCOMPLETE DEVELOPMENT OR GROWTH OF PARTS.

##### OF THE BODY GENERALLY.

Head absent, or rudimentary.  
 Cranium defective.  
 Lower jaw absent or defective.  
 Upper and lower extremities absent.  
 Lower extremities absent.  
 One lower extremity absent.  
 Hands and feet articulated to scapulæ and pelvis.  
 Fingers and toes deficient in number.

##### OF THE NERVOUS SYSTEM.

Brain absent.  
 Brain rudimentary or incompletely developed.  
 Spinal cord absent or imperfect.  
 Continuity of nerves with nerve-centres incomplete.

##### OF THE ORGANS OF SPECIAL SENSE.

Eyes absent.  
 Eyes imperfect.  
 Eyelids incomplete. Eyelids remaining united. (Symblepharon.)  
 External ear absent. Pinna adherent.  
 Meatus externus closed.  
 Internal ear imperfect.  
 Nose absent.

Nose imperfect.  
 Nose resembling a proboscis.

##### OF THE VASCULAR SYSTEM.

Heart absent.  
 Cavities of heart deficient in number.  
 a. One auricle and one ventricle.  
 b. Two auricles and one ventricle.  
 Septa incomplete.  
 a. Auricular.  
 b. Ventricular.  
 Orifices obstructed or imperfect.  
 a. Right auricle, ventricular aperture.  
 b. Pulmonic aperture.  
 c. Left auriculo-ventricular aperture.  
 d. Aortic aperture.  
 Foramen ovale permanently closed.  
 Orifices of aorta and pulmonary artery transferred.  
 Orifices of ascending aorta from left ventricle and of descending aorta from right ventricle through the ductus arteriosus.  
 Commencement of descending aorta contracted or obliterated.  
 Foramen ovale persistent.  
 Ductus arteriosus pervious.  
 Cardiac valves imperfect.  
 Pericardium absent.

##### OF THE RESPIRATORY SYSTEM.

Lung (one or both) absent.

\* To be returned amongst the parasitic diseases of the skin.

Pulmonary lobes deficient in number.  
Larynx and trachea absent or imperfect.

OF THE DIGESTIVE SYSTEM.

Esophagus impervious.  
Intestine impervious, or deficient in various regions.  
Anus impervious.  
Anus in unusual situations.  
Liver preternaturally small.  
Gall-bladder absent.  
Biliary ducts impervious.  
Urachus patent. Vitelline duct patent.

OF THE URINARY SYSTEM.

Kidney (one or both) absent.  
Kidney lobulated.  
Ureters absent or impervious.  
Urachus persistent.

OF THE MALE ORGANS OF GENERATION.

Penis diminutive, resembling clitoris.  
Prepuce abbreviated—elongated.  
Testis (one or both) absent.  
External organs absent.

OF THE FEMALE ORGANS OF GENERATION.

Ovary (one or both) absent.  
Uterus absent.  
Vagina absent.  
Vagina impervious.  
Vagina a cul de sac.  
External organs absent.

MALFORMATIONS RESULTING FROM INCOMPLETE COALESCENCE OF THE LATERAL HALVES OF PARTS WHICH SHOULD BECOME CONJOINED.

A.—ON THE ANTERIOR MEDIAN PLANE.

Fissure of the face.

“ iris. Coloboma.

“ lip.

a. Single harelip.

b. Double harelip.

Fissure of the palate.

a. Hard palate.

b. Soft palate.

“ nose. Naso-buccal fissure.

“ sternum.

“ diaphragm.

“ abdominal walls.

“ pubic symphysis.

“ anterior wall of urinary bladder (with extroversion of posterior half).

Epispadic fissure of the urethra.

Hypospadic fissure of the urethra.

Fissure of the scrotum.

B.—ON THE POSTERIOR MEDIAN PLANE.

Fissure of the skull.

“ spinal column.

Spina bifida.

a. Complete.

b. Partial.

1. Cervical region.

2. Lumbar “

3. Sacral “

Fissure of the spinal cord.

MALFORMATION RESULTING FROM COALESCENCE OF THE LATERAL HALVES OF PARTS WHICH SHOULD REMAIN DISTINCT.

Lower extremities conjoined. Syreniform foetus.

Fingers or toes conjoined.

Monoculus. Cyclops.

Double kidney.

MALFORMATIONS RESULTING FROM THE EXTENSION OF A COMMISSURE BETWEEN THE LATERAL HALVES OF PARTS (CAUSING APPARENT DUPLICATION.)

Double uterus.

Double vagina.

MALFORMATIONS RESULTING FROM REPETITION OR DUPLICATION OF PARTS IN A SINGLE FŒTUS.

Supernumerary fingers and toes.

“ cavities to heart.

“ valves.

MALFORMATIONS RESULTING FROM THE COALESCENCE OF TWO FŒTUSES, OR OF THEIR PARTS.

Fœtus, more or less perfect, contained within another foetus.

Fœtus, more or less perfect, constituting a tumor covered by integument.

Double fœtus.

a. One perfect. The other an appendage.

b. Both more or less perfect.

1. The middle parts united. The upper and lower distinct.

2. The upper parts united. The lower distinct.

3. The lower parts united. The upper distinct.

CONGENITAL DISPLACEMENTS AND UNUSUAL POSITIONS OF PARTS OF THE FŒTUS.

Transposition of viscera.

Hernia or ectopia of the—

brain.

heart.

Hernia or ectopia of the—  
lungs.  
intestines.

*Varieties:*

Through diaphragm. *Syn.*, Diaphragmatic hernia.  
Through abdominal walls. *Syn.*, Abdominal hernia.  
Through umbilicus. *Syn.*, Umbilical hernia.  
Extroversion of posterior wall of bladder.  
Testis retained in abdomen.  
Testis retained in inguinal canal.

DISEASES MANIFESTED AT OR AFTER  
BIRTH.

Prematurely born.  
Stillborn—Asphyxia.  
Atelectasis pulmonum.  
Jaundice.  
Idiotcy.  
Dumbness or deaf-dumbness.  
Congenital cataract.  
Cephalhæmatoma.  
Syphilis.

## PART III.

### THE NATURE OF DISEASES—SPECIAL PATHOLOGY AND THERAPEUTICS.

It is the object of this part to treat of diseases in groups or classes, which possess certain characters or types common to the diseases composing each group; to describe, *Firstly*, The common properties or characters peculiar to the respective classes mentioned in the previous part on systematic medicine; to describe, *Secondly*, The several orders into which these classes of diseases may be subdivided; and, *Thirdly*, To describe in detail the several diseases individually, their *general nature and causes; symptoms, course, and complication; diagnosis, prognosis, and treatment.*

#### CLASS I.

#### ZYMOTIC DISEASES.

---

#### CHAPTER I.

##### GENERAL REMARKS ON THE PATHOLOGY OF ZYMOTIC DISEASES.

THIS class comprises diseases which have been observed to be *epidemic, endemic, and contagious*, and includes *specific fevers, small-pox, plague, influenza, cholera*, and such other diseases as possess the peculiar character in common of suddenly attacking great numbers of people, at intervals, in unfavorable sanitary conditions. In the language of Dr. Farr, the “diseases of this class distinguish one country from another,—one year from another; they have formed epochs in chronology; and, as Niebuhr has shown, have influenced not only the fall of cities, such as Athens and Florence, but of empires: they decimate armies, disable fleets; they take the lives of criminals that justice has not condemned; they redouble the dangers of crowded hospitals; they infest the habitations of the poor, and strike the artisan in his strength down from comfort into helpless poverty; they carry away the infant from the mother’s breast, and the old man at the end of life; but their direst eruptions are excessively fatal to men in the prime and vigor of age. They are emphatically the *morbi populares.*”

The name *Zymotic* (first suggested by Dr. William Farr to desig-



The action of poisons, though definite, is variously limited. Some poisons, for instance, act on one membrane, or on one organ, or on one system of organs; while other poisons extend their action over two or more membranes, or organs, or systems of organs, or even over the whole animal frame. We have examples in aloes and jalap of substances that act mainly upon the mucous membrane of the alimentary canal. In digitalis we have an instance of a medicine that principally acts on the heart, greatly reducing or even stopping its action; while strychnine is an example of a medicine acting on the parts supplied by the spinal cord, producing powerful and sometimes fatal tetanic action of every voluntary muscle in the body.

It is seldom, however, that the action of poisons is limited to one membrane, or organ, or system of organs. The greater number of these noxious agents more usually act on two or more membranes, or organs, or systems of organs. Elaterium, for instance, acts on the mucous membrane of the intestinal canal, and on the kidneys. Tobacco nauseates the stomach, intoxicates the brain, and affects the action of the heart. Antimony has an equally extensive range: it induces cutaneous perspiration, acts cathartically and emetically, and in large doses appears to cause gangrene of the lungs. Alcohol and opium are examples of substances acting still more generally, affecting not only the action or secretion of every organ or tissue of the body, but even in some instances altering their structure. Thus alcohol, in its most limited action, has been shown to cause structural disease of the liver, of the stomach, and of the coats of the arteries. From the circumstance of these substances acting not only generally, but inducing local lesion, they resemble in their effects those of many morbid poisons, as that of typhus fever, of scarlet fever, of small-pox, or of syphilis.

The *second* important law of poisons is, that they lie latent in the system for a period of time which varies in different individuals, before they set up their specific actions. Rhubarb, for instance, produces no immediate result, but lies dormant in the system six or eight hours before its action is sensible on the bowels; opium, in the usual dose, is generally thirty minutes before it subdues the brain. The convulsions from strychnine do not follow till twenty minutes after its administration; and perhaps every substance, except hydrocyanic acid, has a greater or less sensible period of latency.

When a medicine acts on more parts than one, a considerable space of time may elapse after it has affected one organ before it affects another: thus digitalis frequently occasions emesis before it acts on the heart, and the action of mercury on the bowels is frequently sensible for many weeks before the gums and salivary glands are affected. The doctrine of the latency of poisons is indeed so generally admitted that the actual period has been a point on which the condemnation or acquittal of a prisoner tried for murder has turned in our courts of justice, when certain poisons have been supposed to have been given.

The *third* great law of poisons is, that their effects are modified by the dose, the temperament, and the existing state of the consti-





habituate us. On the contrary, each repetition only the more debilitates the constitution, and renders it more susceptible of the action of the poison.

A peculiar existing state of the constitution has also a powerful influence on the action of poisons; and it would seem proved, with some exceptions, that these agents act with an intensity proportioned to the debilitated state of the patient. There is indeed no duty more imperative on the physician than that of adjusting the dose to the strength of the patient; and nothing is more common than to forbear administering a medicine because the patient's strength will not admit of it. As a general principle, therefore, medicines or poisons may be said to act with a power proportionate to the debility of the patient.

Still there are states of disease which render the constitution of the patient, though greatly debilitated, insusceptible to the action of even powerful remedies. Thus, in typhus fever, the patient will often bear a considerable quantity of vinous stimuli without being affected by it. In tetanus, or hydrophobia, no quantity of opium will tranquillize the symptoms or procure sleep. Fallopius mentions a singular instance of the constitution being armed against the action of a poison. He states that in his day a criminal was given up to himself and other anatomists, to be put to death in any manner they might think proper. To this man, therefore, they administered two drachms of opium, but, laboring under a quartan ague, and the fit just coming on, the "opium was hindered of its effect." The man, therefore, having survived this dose, begged that he might take a similar quantity, earnestly entreating, if he escaped, that he might be pardoned. The same dose was repeated, but it was in the interval of the attacks, and the man died.

The experiments of Majendie may be referred to as affording many curious proofs of the state of the constitution in accelerating or retarding the action of poisons. He has shown that if a poison be introduced into the system, of such potency as usually will destroy life in two minutes, on bleeding the animal the same result will follow in half a minute, or in one-fourth of the time; and this experiment has often been repeated. Majendie also brought to light the curious fact, that if, after having poisoned the animal, and even after the poison has begun to act, we inject an aqueous fluid into its veins in such quantity as to cause an artificial plethora, as long as this artificial plethora can be maintained, the action of the poison is superseded. No sooner, however, does the plethora cease, from the general effusion of fluid into every cavity of the body, than the poison acts in the usual time, and with even perhaps more than its accustomed severity.

Mr. Hunter thought that no two poisons could *coexist* in the same system together, or that, coexisting, they could not set up their specific actions at the same time. This hypothesis, however, is unquestionably erroneous; for we constantly see opium and digitalis, jalap and mercury, as well as many other combinations of medicines, producing their respective effects in the same system, and at the same time, by accelerating or retarding each other's



membranes of the joints and the abdomen. The *paludal* and the *syphilitic* poisons have a still more extensive range, hardly any organ or tissue of the body being exempt from the destructive ravages of these poisons.

Morbid, like other poisons, have their periods of latency; and, generally speaking, a much longer time elapses before their specific actions come into operation than with medicinal substances. The virus of the *natural small-pox* lies dormant from sixteen to twenty days before it produces any constitutional disturbance; and a still further period elapses, of three or four days, before the specific eruption appears on the skin. The poison of *scarlatina* lies latent from seven to ten days after exposure to the contagion; that of *measles* from ten to fourteen; while the poison of *paludal fever* has been said to lie dormant for a twelvemonth, and that of *hydrophobia* for a still longer time. These are examples of periods of latency far beyond anything that has been observed in the action of medicinal substances; and *syphilis* in its remote effects upon the organs and the constitution generally is still more remarkable.

When morbid poisons act on more tissues or organs than one, their actions are sometimes simultaneous, but more commonly they are consecutive, and frequently long intervals of time elapse between each successive attack. Thus, the poison of *typhus* and *enteric fever* may affect the lungs, the membranes of the brain, and the mucous membrane of the alimentary canal, and all these may be attacked contemporaneously, but more often consecutively; or first on the alimentary canal, then on the brain, and lastly on the lungs, several days elapsing between each successive affection.

It occasionally happens that morbid poisons which usually act on a plurality of membranes, exhaust themselves on one or more without affecting others. In *scarlatina simplex* the poison sometimes exhausts itself entirely on the skin, without affecting either the mucous or serous membranes of the body. The *rubeola sine catarrho* is a similar example of the poison exhausting itself on the skin. In *intermittent fever*, when the dose of the poison is limited, and the disease properly treated, it is seldom that a lesion occurs in any organ or tissue; yet, left to run a slow course, with constant exposure to the poison, scarcely any organ or tissue would escape being affected and its function impaired.

Sometimes, when the morbid poison acts on many membranes, the usual order of attack is inverted. In *scarlet fever* the affection of the skin may precede that of the throat, or the reverse may take place.

It has been seen that the period of latency of medicinal substances having passed over, the effects vary in a considerable degree, according to the dose, temperament, or present state of the constitution of the patient. With respect to the dose of a morbid poison, we rarely possess any direct measure of its strength. The *paludal* poison of tropical climates, to which *malarious fevers* are due, unquestionably greatly exceeds in intensity that of more temperate climates, and its effects are proportionally marked. Thus, in the West Indies, the severe remittent fevers occur with hardly a trace



**Peculiarities in the action of Poisons which induce Zymotic Diseases.**—The principal points in which the effects of poisons which induce Zymotic diseases agree with those of poisons generally having been stated, it will now be necessary to state those circumstances in which they principally differ. Many medicinal poisons have the property of accumulating in the system, and acting with an intensity proportioned, not to the last dose, but to the aggregate of the whole quantity that has been administered. Thus the last few minims of digitalis may stop the action of the heart, or the last few grains of mercury salivate the patient, or the last minute dose of strychnine become fatal. There is, however, no well-authenticated fact which can be arranged under this law in the whole circle of morbid poisons, except, perhaps, the cumulative and persistent pernicious action of paludal malaria. The actual quantity required to establish disease, according to the experiments of Dr. Fordyce, is probably extremely small. That physician, in the hopes of mitigating the small-pox, inoculated with virus greatly diluted; and although the disease was not always produced, yet when produced, it assumed every form, character, and degree of severity that small-pox has ever been known to assume.

The puerperal female is not only highly susceptible of poisons of the Zymotic kind, but she is proved to favor their further development; and forms of puerperal fever seem capable of generation by *materies morbi* of a kind other than that which might be considered peculiar to it. It is a well-known fact, unhappily not of rare occurrence, that a medical practitioner or a nurse from a case of puerperal fever going to attend on other cases of labor, the chances are that these will be attacked with the disease. Further, the practitioner or nurse may go to cases of labor from attendance on a case of scarlatina, typhus, erysipelas, or small-pox, and the parturient patients may then become the victims of puerperal fever. In the Vienna Lying-in Hospital it is on record that a mortality of 400 to 500 in an average of 3000 deliveries per annum appeared traceable to the introduction of cadaveric matters, through the uncleanness of the attending students; these matters, being especially potent when derived from the bodies of those who have died from the adynamic forms of Zymotic disease. Students of practical midwifery should bear in mind this fact. They ought not to attend cases of labor while they are also engaged with practical anatomy in the dissecting-room.

Another peculiar law of morbid poisons, and one wholly unknown in medicinal substances, is the faculty which the human body possesses of generating to an immense extent a poison of the same nature as that by which the disease was originally produced. A quantity of small-pox matter not so big as a pin's head will produce many thousand pustules, each containing fifty times as much pestilent matter as was originally inserted; and, moreover, the blood and all the secretions of the body are equally infected with the matter of the pustules. The miasmata from one child laboring under whooping-cough are sufficient to infect a whole city.

There is still perhaps a more remarkable law of morbid poisons,



period than twenty-five seconds, though the phenomena of poisoning did not occur till several minutes later.

The experiments of Fontana had shown (supposing a poison to be introduced into the veins) that all the phenomena of poisoning were accounted for; but still it might be said that to prove the fact of absorption something was wanting in strict demonstration. For the further prosecution of this subject we are indebted to Segalas, who showed that if the arteries and veins of the mesentery of a dog be tied, a quick-acting poison would lie in harmless contact with the corresponding portion of the intestine for many hours; but no sooner were these ligatures removed than poisoning took place in a few minutes. Majendie has carried this proof, of the veins absorbing, even still further. He amputated the leg of a dog, having first introduced a portion of quill into the femoral artery and vein, in such a manner that, on dividing these vessels, the leg hung connected with the trunk solely by means of the quill, all continuity by means of the solids being cut off. The poison was now introduced into the tissues of the paw, and in four minutes the animal was under its influence.

By these experiments it is believed that Fontana, Segalas, and Majendie have completely demonstrated the absorption of poisons by the veins, and consequently of their circulating with the blood; and that no doubt may remain on the subject, modern chemistry has demonstrated the actual presence of many medicinal substances either in the blood itself or in the secretions formed from it. Thus, after the free use of soda, large quantities of uncombined alkali have been found in the serum. Alcohol has been obtained by distillation from the blood; while iodine, rhubarb, the nitrate of potash, and a large number of other substances taken into the stomach have been found in the urine. It follows, then, that poisons are absorbed and mingled with the blood, and are conveyed directly to the parts on which they act, passing with impunity over others for which they have no affinity.

The fact of morbid poisons in like manner being absorbed, and mingling with the blood, has been shown by many Continental writers; but perhaps the experiment made by Professor Coleman is the most satisfactory. "I have produced the disease (the glanders) by first removing the healthy blood from an ass, until the animal was nearly exhausted, and then transfusing from a glandered horse blood from the carotid artery into the jugular vein. The glanders in the ass was rapid in its progress, violent in degree, and from this animal I afterwards produced both glanders and farcy." Scarlatina, measles, and syphilis have now been produced by inoculation *from the blood* of patients laboring under these diseases.

The circumstance of the presence of a poison in the blood is supposed by Andral to produce, besides its toxic states, certain alterations in its physical condition. Thus a specific cause has a tendency to destroy or reduce the quantity of fibrine in the blood, which he has found in some instances to be only one part in a thousand. Hence he adds, whatever may be the nature of the specific pyrexia, the blood always exhibits the following characters, whether it be





ORDER 2. ENTHETIC DISEASES—*Enthetici*.

ORDER 3. DIETIC DISEASES—*Dietici*.

ORDER 4. PARASITIC DISEASES—*Parasitici*.

---

## CHAPTER II.

### PATHOLOGY OF THE Miasmatic Order of Zymotic Diseases.

THE diseases to be described under this order acknowledge at least three sources or modes of origin; while they are all mainly propagated, disseminated, communicated, or diffused through the agency of contaminated persons, food, water, or other agents, or through infected air. The poisons, miasms, gases, germs, active principles, or morbid agents may be arranged under three classes, namely,—1st. Paludal malarious poison; 2d. Animal malaria poison; 3d. Specific disease poisons. The diseases they engender are attended by a febrile state, which may assume various forms or types.

**Paludal Malarious Poison.**—This poison arises from marshy land in particular conditions, such as decomposition, under the influence of partial moisture, and of heat above 60° Fahr. If the land is perfectly dry or perfectly flooded the poison is not generated. It is a material poison. It may be wafted along with the wind, and so induce fever at a distance from the place where the poison is generated. It may also be intercepted by a belt of trees. It appears to be most intense near the surface of the ground. The diseases usually attributed to this endemic source, and which were formerly so destructive, have almost disappeared from this country. The reason of this may fairly be ascribed to the improved drainage both of the towns and of the agricultural districts. The fact may be proved, did space permit; and the practical inference leads one to hope for still more immunity from diseases arising from this source, if the “proper authorities” direct further efforts in this direction. “Within the last half century land-draining and town-sewering have ripened into sciences. From rude beginnings, insignificant in extent, and often injurious in their effects in the first instance, they have become of the first importance. Land has, in many instances, doubled in value; and town-sewering, with other social regulations, have not unfrequently prolonged human life from five to fifty per cent., as compared with previous rates in the same district.” “Agues (and malarious cachexiæ) are reduced. Since 1840 an annual mortality in English towns of 44 in 1000 has been reduced to 27; an annual mortality of 30 has been reduced to 20, and even as low as 15; and human life has now more value in England than in any other country in the world—a result entirely due to better sanitary arrangements” (Rawlinson, *Journal of Society of Arts*, March 21, 1862, vol. x, p. 276).



and the ingestion of alcoholic drinks. Each and all of these causes tend to induce morbid conditions of the blood, a tendency to putrescence, and a condition of ill-health of the body.

**Specific Disease-Poisons.**—The matter by which the specific miasmatic diseases are communicated and propagated is solely derived from the body of the similarly diseased human or animal being; for there, during the course of the specific disease, is the soil in which the specific poison is bred, to multiply and propagate its kind. It is not yet clearly established how far the bodies of animals may not be a soil for the propagation of diseases communicable to man. (See the Sections on "Small-pox," and "Cow-pox.")

The diseases of the lower animals are not sufficiently studied by us. The diseases of plants are almost entirely neglected. Yet it is clear that until all these have been studied, and some steps taken to generalize them, every conclusion in pathology regarding the nature of the propagation and dissemination of specific miasmatic, and even of parasitic, dietic, and enthetic diseases, must be the result of a limited experience from a limited field of observation. How do we know that the blights of plants, or the cause of them, are not communicable to animals and to man? We know how intimately related the diseases of man and animals are with famines and unwholesome food; and of famines with the diseases of vegetable and animal life, as much as with the destruction and loss of food.

Dr. Carter, of Bombay, has shown that there is in India a very singular, and although strictly endemic disease, yet a very prevalent one, which occurs in the hands and feet, especially the latter, and which it is probable is really of the nature of a "blight," in so far as it is owing to the implantation in the tissues of "sporules or germs," which in the progress of development commit irremediable ravages on the affected parts, leading ultimately to entire disorganization of the tissues. It is known as the "fungus disease of India," originally described by Dr. Carter in the *Transactions of the Medical and Physical Society of Bombay*, No. 6, new series for 1860. (See the account of "Parasitic Diseases" at the end of this volume.)

On the relations between the diseases of man and animals, and especially in connection with food, the reader is referred to a series of papers by the author, in the *Medical Times and Gazette* for 1857.

Dr. William Budd, of Bristol, has also recently directed attention to the occurrence of malignant pustule in *England*, in a paper read at the great meeting of the British Medical Association in London, in August, 1862. He has shown that the disease has not been so uncommon in England as had been supposed—that it is common and very fatal to oxen and sheep in this country—that in man and in sheep the disease is identical—that it is communicable to man by direct inoculation, and also by eating the flesh of the animals affected—that it may be conveyed and disseminated by the bites of insects, such as gnats—and that the disease may be recommunicated from man to animals (*Brit. Med. Journal*, January 24, 1863).

There are some peculiar and characteristic features especially



vailing disease only begins to prevail in the new locality after it has already died out in the old. 4th. One element remains constant in the history of endemic influence, and that is *the specific morbid poison* which is the origin of each case. It is susceptible of transmission from place to place, gathering strength as it proceeds, again to die out or become dormant, so that its track is with difficulty followed or traced out. 5th. In large cities such specific poisons are always more or less active, and their diseases always present; but in the country districts they only now and then occur. The occurrence of long intervals of rural exemption is not traceable to any feebleness of the poison to act; for when the disease does become developed in these places, the ratio of persons or of animals attacked is incomparably greater than is ever seen in cities under like circumstances (see Professor Acland's account of the fever in Great Horwood, in 1857-58; and Dr. William Budd, of Clifton, regarding fever at North Tawton; and his most instructive little book *On the Propagation of Typhoid Fever*). 6th. In large towns the sewers are constantly charged with the *materies morbi* of specific diseases always abounding in towns. In small villages, and other places where no sewers exist, the air only may be infected, or the water contaminated, by the direct or indirect importation of cases of specific disease or their equivalents—the poison itself—so that the organic impurities, the dung-heaps, the open soil which surrounds the dwellings of the patients, the cess-pools, and the privies common to several houses, gradually but eventually become impregnated with the specific poison of the disease. Thus the atmosphere of the village may become incomparably more virulent than the atmosphere of the sick-chamber itself. Hence the rapid epidemic spread of the miasmatic diseases in the limited space of rural villages; and which gives rise to the popular error, that such diseases are invariably contagious in country places, and only rarely so, or by exception, in cities or large towns. 7th. All these specific diseases multiply their kind after similar modes of propagation. 8th. All of them establish a constant series of morbid changes and lesions, and always issue in the reproduction of its own specific germ, miasm, gas, morbid poison, or active principle by which it propagates its kind. Thus small-pox propagates small-pox; measles multiplies measles; scarlatina reproduces scarlatina; typhoid fever breeds typhoid fever; typhus, typhus; and so on. In the terse language of Dr. William Budd,—“What small-pox and measles were in the Arab in the days of Rhazes, they still are in the London Cockney of our own time. What they are in the London Cockney, they are in the wild Indian of the North American prairie, and in the Negro of the Gold Coast. To all the other specific communicable diseases, as far as our records go, the same remark applies. In races the most diverse, under climates the most various, age after age, through endless generations of man, these diseases pass down through the human body (sometimes through animals—*e. g.*, ovine small-pox?), perpetuating their own kind, and each maintaining its separate identity by marks as specific as those which distinguish the asp from the adder or the hemlock from the poppy.” Such being the case, it is difficult to conceive (as Drs. Watson and William Budd



is known to us by inference only. Again, we know that ample provision is made and ways are open for the dissemination of the active agent of propagation in a thousand unseen modes, so that it is obvious that the precise sort of infection and its track must often baffle the wisdom of man to discover or trace out.

Cases thus constantly arise which appear to give countenance to the belief that the disease has had a spontaneous origin—*sporadic*, as it is termed. Numerous cases of small-pox occur which can never be traced to their source, or to communication with persons similarly diseased; yet the history of small-pox is decisive against the notion of its spontaneous origin; and if of small-pox, so for all the other specific Zymotic diseases of the same nature. Dr. Watson has well observed that “the small-pox never occurs except from contagion. *It was quite unknown in Europe till the beginning of the eighth century.* No mention of any such malady is to be found in the Greek or Roman authors of antiquity. Now, whatever may have been the deficiencies of the ancient physicians, they were excellent observers and capital describers of disease; and it is impossible that a disease so diffusive, and marked by characters so definite and conspicuous, should have escaped their notice, or have been obscurely portrayed (if known) in their writings. On the other hand, Mr. Moore, in his learned and interesting *History of Small-Pox*, has shown that it prevailed in China and Hindostan from a very early period—even more than a thousand years before the time of our Saviour. That it did not sooner extend westward into Persia, and thence into Greece, may be attributed partly to the horror which the disease everywhere inspired, and the attempts that were subsequently made to check its progress, by prohibiting all communication with the sick, partly to the limited intercourse which then took place among the Eastern nations, but principally to the peculiar situation of the regions through which the infection was diffused, separated as they are from the rest of the world by immense deserts and by the ocean” (Watson, *Lectures on the Practice of Physic*, 3d edition, vol. ii, p. 709). “If anything were wanting,” writes Dr. Budd, “to show what is the true inference to be drawn from these events, it would be found in the fact that, *once imported into the West*, it spread with the most fearful rapidity and havoc; and that while almost all men are prone to take the disorder, large portions of the world have remained for centuries exempt from it, until at length it was imported, and that then it infallibly diffused and established itself in those parts. In this country the (endemic) conditions for the spread of the disease existed in the most intense degree, as was shown by the event when the disease was once introduced. The long lapse of ages during which we remained entirely free from small-pox showed, with equal clearness, that, until this introduction occurred, all the conditions favorable to the development of small-pox were powerless to cause a single case. The spectacle witnessed in Europe was repeated over again in the Western World in a still more striking way. Our knowledge of the events here is precise and sure. There was no small-pox in the New World before its discovery by Columbus, in 1492. In 1517 the disease was imported into St. Domingo. Three





ditions, tending in certain localities to determine a specific decomposition of excrement, communicable to other organic substances and infecting the air, is an essential element in an epidemic period.

The most recent speculation regards the discovery of a peculiar atmospheric condition, ascribed to a principle called *ozone* or *osmazone* (ὀσων, stink, or ὀσμή, smell), of which, as yet, we know nothing definite, although many subtle instruments and apparatus are in use to detect and measure the amount of this principle in the air.

A careful study of the effects of the *epidemic influence* appears to warrant the enunciation of certain laws which seem to regulate its operations. These laws may thus be condensed:

**Laws of Epidemic Influence.**—(1.) This influence frequently predisposes to diseases, apparently independently of any other known cause, as in the case of influenza and cholera. It makes itself manifest by appearing to give increased energy to causes which produce particular diseases; so that small-pox, scarlatina, typhus, and the like, sometimes rage with great violence as epidemics. It also appears to predispose to new and anomalous forms of disease, as witnessed in the furunculoid epidemic which recently prevailed both in Europe and America, from 1849 till 1852. (2.) Sometimes the *epidemic influence* manifests itself by a certain type or direction which existing diseases appear to take. Thus, at one period diseases take a low, or what is called a *typhoid* type, so that depletion is not tolerated; at another time an *inflammatory* tendency predominates, and antiphlogistic treatment is required. At one period there is a tendency in disease to complicate its course by a disposition to affect particular organs. At one time head affections predominate; at another time affections of the chest, or of the alimentary canal, complicate the course of a prevailing disease. Consequently the same disease may demand very different, and even opposite, modes of *management*. (3.) During epidemics other diseases are apt to assume more or less of the prevailing epidemic features. Thus, when cholera prevails, looseness of the bowels often complicates the course of other affections. When influenza prevails, catarrhal complications increase the danger of other diseases. Ill-health of any kind, therefore, favors the action of the epidemic influence. (4.) Some change in the character of prevailing diseases of a constant and recurring kind often indicates the approach of an epidemic and the prevalence of the epidemic influence. (5.) The first effects of the *epidemic influence* are usually the most violent and marked, and the cases of the epidemic disease become mild as the *epidemic influence* passes away. (6.) The *epidemic influence* sometimes disappears entirely after a short prevalence; sometimes continues, with irregular intermissions, for two, three, four, or even six years, or longer. Influenza and cholera are examples. (7.) An epidemic tendency, after continuing for several years, may give place to one of a different kind, which, in its turn, may again give place to the first. *Malarious fevers, yellow fever, and typhus*, illustrate this in America. The eruptive affections seem to run in somewhat similar cycles. After the introduction of vaccination the *small-pox* seemed for many years to be almost entirely subdued; but more recently again the disease has seldom been en-



case. Next to large towns, the health of the Army is of the greatest importance, especially when we consider the tendency that exists to a high rate of mortality in that service. In the military age (which is the age between eighteen and forty) the mortality of the general population in England is less than one per cent. per annum. The mortality of the British army is much above this. On Home service it has had a mortality double that of the civil population at the corresponding ages; and seven-ninths of the entire mortality among the infantry of the line has arisen from diseases of the Zymotic class. Disease and mortality are much greater during campaigns, when more than twenty-two per cent. are constantly on the sick list. The causes of high rates of mortality require constant investigation, by carefully observing, recording, and comparing the facts over a sufficiently large area; thus arriving at certainty as to the causes, and whether they can be mitigated or removed.

An observation of great interest in connection with *animal malaria poison*, as well as with *epidemic influences*, may be appropriately referred to here. It seems clearly proven, especially by the valuable and decisive observations of Dr. William Budd, of Bristol, that the communicable poisons of typhoid fever and of cholera are capable of being imported or carried from place to place by persons who have the disease. Dr. Budd's history of the North Tawton fever and its offshoots (*Lancet*, July 9, 1860) is most conclusive on this point. His arguments are also cogent to the general effect that *specially* the bowel discharges are *means* by which a patient, whether migrating or stationary, can be instrumental in disseminating typhoid fever and cholera. Mr. Simon makes the important remark, however, that these bowel discharges may not be the *sole means* of multiplying and disseminating these diseases; although, provisionally, the conclusions of Dr. Budd must be acted upon in their present unqualified form; while it is of the greatest practical importance to learn, as exactly as possible, whether it is in all states of the disease, and under all circumstances, that the bowel discharges of typhoid fever and cholera can communicate and multiply the means of dissemination. In illustration of such possible contingent results, Mr. Simon refers to some interesting and important experiments made in 1854 by Professor Thiersch, of Erlangen. These experiments seemed to show that cholera evacuations, *in the course of their decomposition*, either acquire the power of communicating or multiplying their specific poison, or that the specific poison inherent in them becomes intensified by decomposition (Zymosis?). That the decomposition or change may begin even in the bowels, after the secretion and accumulation of the material in them, as well as in cesspools, seems to be possible; and perhaps, as Mr. Simon justly remarks, may furnish an explanation of the many cases in which human intercourse has apparently disseminated the disease. For, according to the observations of Professor Pettenkofer at Munich, and Professor Acland at Oxford, it would seem that during cholera periods the immigration of persons suffering apparently only from diarrhœa has been followed by outbreaks of cholera in places previously uninfected; and Professor Pettenkofer ascribes this fact to an



contained in the following statement, namely: That it is possible to extinguish the greater number of epidemic diseases, however intense or abundant may be the atmospheric or other agencies which constitute their potential causes, by remembering,—(1.) That the living body of the diseased persons is the soil on which the communicable disease breeds the poison by which the specific disease is multiplied and propagated: (2.) That excretions from an infected person, especially such excretions as are immediately related to or flow from parts affected with specific lesions, probably contain the most active elements of the specific poison by which the disease may be disseminated: (3.) That such active elements, germs, poisons, miasms, gases, or noxious agents may contaminate the drinking-waters of a district, or may infect the atmosphere, or lie dormant for variable and unknown periods of time, just as seeds dry up and preserve their vital properties: (4.) To follow out zealously the hygienic measures which flow from these statements, and so prevent the propagation of specific diseases: (5.) To preserve as much as possible the blood of every individual in that state which shall prevent these poisons from finding the conditions of their development within the body: (6.) That these ends are to be attained on the one hand by preventing the production of fermentable matter in or out of the body; and on the other hand by promoting its removal and chemical destruction or decomposition, when it is inevitably generated, and by a free supply of pure air, and by the reduction of that air to the lowest temperature at which the condition of the individuals will allow it to be safely inhaled. Preventive measures based upon these principles are of the utmost importance, so much so that the most eminent members of the medical profession in London and elsewhere concur in the views and opinions of Dr. William Budd, unanimously cherishing the maxim, that, “except under the pressure of great military straits, no army ought ever to suffer on a large scale from this great group of communicable diseases, and especially such as are disseminated by intestinal discharges.”

The following detail of proceedings advisable to be taken in places attacked or threatened by epidemic diseases are given mainly from a memorandum drawn up by John Simon, Esq., the Medical Officer of the Privy Council, and published in his *Third Report on the Public Health in England in 1860*:

1. Wherever there is prevalence or threatening of cholera, diphtheria, typhus, or any other epidemic disease, it is of more than common importance that the powers conferred by the Nuisances Removal Acts, and by various other laws for the protection of the Public Health, be vigorously, but at the same judiciously exercised by those in whom they are vested; and with regard to armies, that the instructions relative to the guidance of the Medical Officer in sanitary matters, contained in the *Army Regulations*, be duly carried out, on the principle that the executive should act under authority, in order to carry out the required measures efficiently.

2. If the danger be considerable, it will be expedient that the local authorities in civil life, and the commanding officers of armies, brigades, divisions, and regiments, in military life, avail themselves,



efficient ventilation even at some real or imaginary expense of comfort.

10. The cleanest domestic habits should be enjoined. Refuse matters should never be suffered to remain or to linger within the dwelling, hospital, barrack-room, or hut. Such refuse must *at once* be removed, and at once disposed of, or cast into the receptacle provided for it. All things or utensils which have to be disinfected or cleansed should always be disinfected or cleansed *without delay*.

11. With regard to material substances discharged or separated from the bodies of the sick, special precautions of cleanliness and disinfection are necessary. Among discharges or substances separated from the body which it is proper to treat as capable of communicating disease, are those which come, in cases of small-pox, from the affected skin; in cases of cholera and typhoid fever, from the intestinal canal; in cases of diphtheria and scarlatina maligna, from the nose and throat, and the exhalations from the skin and the lungs saturating clothes; likewise, in cases of eruptive fevers, measles, scarlatina, r  theln, typhus, and the like, the general exhalations of the sick, and especially so of the convalescing, probably in connection with the desquamation of the skin. The caution which is necessary with regard to such matters must of course extend to whatever may be imbued with them; so that bedding, clothing, towels, and other articles which have been in use by the sick, do not become sources of mischief, either in the house to which they belong, or in houses to which they are conveyed. Moreover, in typhoid fever and cholera, the evacuations should be regarded as capable of communicating a similarly specific and infectious property to any night-soil with which they may be mingled in privies, drains, or cesspools (THIERSCH). This danger of multiplying the sources of communicating disease must be guarded against by the chemical destruction, decomposition, or disinfection of all the intestinal evacuations as soon as they are passed from the bowels, and certainly before they are thrown away, and so "let loose upon the world." Above all, they must never be cast where they can run or soak into sources of drinking-water.

12. All reasonable care should be taken not to disseminate disease by the unnecessary association of persons suffering from the specific communicable diseases, either with healthy persons, or in wards of hospitals where patients suffering with other diseases are being treated. This care is requisite, not only with regard to the sick-house, ward, hospital, or ship, but likewise with regard to day-schools, places of public resort, courts of justice, and other places where members of many different households are accustomed to meet.

13. Where dangerous conditions of residence cannot be promptly remedied, it will be best that the inmates, while unattacked by disease, remove to some safer lodging. If disease begins in houses where the sick person cannot be rightly circumstanced and tended, medical advice ought to decide on the propriety or fitness of removing him to an infirmary or hospital. In extreme cases, special in-





discharges from the patient immediately removed, and the utensils washed.

3. Nurses and attendants ought to endeavor to avoid the patient's breath and the vapor from the discharges.

4. Visitors must not go near to the sick, nor remain with them longer than is absolutely necessary: they should not swallow their spittle, but clean the mouth and nostrils when they leave the room.

5. No dependence must be placed on vinegar, camphor, or other supposed preventives, which, without attention to cleanliness and admission of fresh air, are not only useless, but by their strong smell render it impossible to perceive when the room is filled with bad air or noxious vapors.

**Processes of Disinfection.**—These processes have been recommended by Professor Miller, of King's College, London. They cannot supply the place of cleanliness, ventilation, and drainage. They are artificial, and are used for exceptional purposes, the great natural disinfectant being *fresh air*, abundantly and uninterruptedly supplied.

1. For purposes of artificial disinfection, the agents which most commonly prove useful are, chloride of lime, quicklime, Condyl's manganic compounds, and carbolic acid. Metallic salts, especially perchloride of iron, sulphate of iron, and chloride of zinc, are under some circumstances applicable. In certain cases chlorine gas or sulphurous acid gas may advantageously be used; and in certain other cases powdered charcoal or fresh earth.

2. If perchloride of iron or chloride of zinc be used, the common concentrated solution may be diluted with eight or ten times its bulk of water. Sulphate of iron or chloride of lime may be used in the preparation of a pound to a gallon of water, taking care that the water completely dissolves the sulphate of iron or has the chloride of lime thoroughly mixed with it. Condyl's stronger fluid (red) may be diluted with fifty times its bulk of water; his weaker fluid (green) with thirty times its bulk of water. Where the matters requiring to be disinfected are matters having an offensive smell, the disinfectant should be used till the smell has entirely ceased.

3. In the *ordinary emptying of privies or cesspools*, use may be made of perchloride of iron, of chloride of zinc, or of sulphate of iron. But where disease is present, it is best to use chloride of lime or Condyl's fluid. Where it is desirable to disinfect before throwing away the evacuations from the bowels of persons suffering from certain diseases, the disinfectant should be put into the night-stool or bed-pan when about to be used by the patient.

4. *Heaps of manure* or of other *filth*, if it be impossible or inexpedient to remove them, should be covered to the depth of two or three inches with a layer of freshly burnt *vegetable* charcoal in powder. Freshly burnt lime may be used in the same way, but is less effectual than charcoal. If neither charcoal nor lime be at hand, the filth should be covered with a layer, some inches thick, of clean dry earth.

5. *Earth near dwellings*, if it has become offensive or foul by the



*mucous surfaces, with other concomitant and occasionally succeeding affections. The eruption on the skin passes through the stages of pimple, vesicle, pustule, scab, and leaves marks or cicatrices on its site. The disease runs a definite course, and as a rule, exhausts the susceptibility of the constitution to another attack.*

**Pathology.**—The theory regarding the development of small-pox is, that a specific poison is absorbed and infects the blood, and after a given period of latency gives rise to “primary fever,” which lasts from two to four days, till the eruption appears, when the fever for the most part *remits*. The secondary or specific action of the poison of small-pox makes itself obvious by an eruption on the skin, and also sometimes on the mucous membrane of the eyes, nose, mouth, fauces, and great intestine. The eruption runs a given course—namely, *pimple, vesicle, and pustule*—and when fully out, or at its height, the febrile phenomena, which had remitted, return, and give rise to what is termed the *secondary fever*. The occasionally succeeding morbid conditions are inflammation of the various tissues of the lungs, of the urinary organs, and, lastly, of the areolar tissue of the body generally, which often becomes the seat of an endless number of abscesses.

The occurrence of fever preceding the secondary or specific actions of the poison, or the appearance of the eruption, has scarcely an exception, and, indeed, in some instances it has been of so severe a character as to have destroyed the patient on the first onset. The remission or subsidence of the fever is constant in mild cases, but in the severer forms of the confluent small-pox it sometimes runs on, and is constant. The recurrence of the “secondary fever,” and the exacerbation of the fever in severe cases at the time of the maturation of the pock, is also constant. The cause of this secondary attack has long been a difficulty in the pathology of small-pox. Some attribute the fever to the specific nature of the disease, while others consider it to result from the maturation of the pustules, and to be a *suppurative fever*—*symptomatic*, and dependent upon the local affection.

Another constant phenomenon in the development of small-pox is, that the secondary actions of the poison occasion a peculiar eruption. There are a few rare exceptions, which constitute a variety of small-pox sometimes noticed as the “*variola sine eruptione*.” The affection of the mucous membranes is often wanting in mild cases, though rarely absent in severe ones. The poison is also apt to set up many tertiary actions, as inflammation of the lungs, of the urinary organs, of the eye, and of the areolar tissue. Generally it may be mentioned that the state and appearance of the eruption depends in a great measure upon the type and character of the fever, while the type and character of the fever may be modified by the organic functions and condition of the blood, especially as induced by vaccination.

The development of small-pox is traceable through certain stages, namely, 1st. The stage or period of incubation; 2d. The febrile stage, or period of primary fever; 3d. The exudative stage, or



the cutis are the cells and tissue of the *rete mucosum* (GRUBY, GLUGE, RAYER, GUSTAV. SIMON, besides other observers of more early date). Will not some delicate process of organic analysis tell us what the *active principle* of the specific virus of small-pox is,—if it be capable of being so determined? While the maturation of the vesicle into a pustule is going on, a damask red areola forms around each pustule; and as the vesicle fills, the whole face swells, and often to so great a degree that the eyelids are closed. While the maturation is complete, the “*bride*,” which bound down the centre of the vesicle, ruptures, and the pustule now becomes *spheroidal* or *acuminated*. About the eighth day of the eruption a dark spot is seen on the top of each pustule. At that spot the cuticle ruptures, allowing matter to exude, which concretes into a scab or crust; and during this process the pustule shrivels and dries up. The crust is detached between the eleventh and fourteenth days, leaving the cutis beneath of a dark reddish-brown hue—a discoloration which lasts many days, or even weeks. On the face, however, the pustule often penetrates or burrows, so as to cause ulceration of the *rete mucosum*, leaving a permanent cicatrix in the form of a depression or “pit.” The cicatrix thus formed, though at first of a reddish-brown, ultimately becomes of a dead white color.

The small-pox eruption does not appear over the whole body at once, but appears in three successive crops. The first crop covers the face, neck, and upper extremities, the second the trunk, while the third appears on the lower extremities. There is usually an interval of several hours between each crop; and the later the pustules are in appearing on the trunk and lower extremities than on the face and neck, by so much the later they are in maturing, and in disappearing from those parts. When the eruption on the face is declining, that upon the extremities has scarcely yet arrived at its height, so that the hands and feet are then considerably swollen. This is to be regarded as a favorable sign, in so far as it indicates a certain vigor of constitution.

The number of pustules is very various, sometimes not exceeding five or six over the whole body, more commonly from one to three hundred, and occasionally amounting to several thousands. It has been calculated, if ten thousand pustules be counted on the body, that two thousand at least will be found on the face; and accordingly, the number of pustules on the face being in proportion, those on the other parts of the body furnish a fair estimate of the extent of the disease, and of the danger of the patient.

The pustule is subject to many irregularities, both as to its form and course; which gives rise to two very marked varieties of the disease, namely, the *confluent* and the *horn* small-pox. The *confluent small-pox* differs from the *distinct small-pox* in the pimples being small, less prominent, and so numerous that even on the first appearance of the eruption there is hardly any distinct separation between them. The vesicles which form on their apices appear earlier, and their diameters increase more irregularly than in the distinct forms, and often they run one into the other. The pustules, likewise, which are confluent, either remain flat, and do not



urine,—and hence the “*variolæ sine eruptione*” of authors,—which, when it occurs in the present day, is more usually regarded as a modification of small-pox, probably depending on the influence of vaccination.

**Symptoms of the Distinct Small-Pox.**—The symptoms of *variolæ discretæ*, or of distinct small-pox, may be divided into *four stages*. The *first stage* comprises the period of incubation or of latency—a period of time which varies according as the poison has been introduced by the mucous or cutaneous tissues. In the former case, or in natural small-pox, for example, the more usual time of latency is from ten to sixteen days; while in the inoculated small-pox the period of latency is from seven to nine days, the extremes, taking both forms of the disease, being from five to twenty-three days. Bärensprung, of Berlin, has lately recorded a most interesting fact, which demonstrates, in a more striking and definite manner, the period of latency; and which appears to be similar in persons who have been vaccinated and in those who have not. He observed seven cases of small-pox, *all* of which were infected from the same source on the same day. In *all* of them the outbreak occurred between the thirteenth and fourteenth day. Some of them were vaccinated and some were not (*Annalen des Charite Kranken*, vol. xix, p. 103). The *second stage* comprises the primary fever, which commences with the disease, and terminates with the appearance of the eruption. The *third stage* commences with the eruption, and terminates with the appearance of the secondary fever. The *fourth stage* commences with the secondary fever, and includes all the subsequent phenomena.

In the adult the symptoms of the second stage are mainly to be distinguished from those of the first stage of typhus, or other febrile affections, by the characteristic ranges of temperature. There is, however, a great tendency to vomiting, and to pain in the back, and the brain is oppressed, as indicated by drowsiness, stupor, or coma, followed occasionally by convulsions. The ordinary duration of this fever is four days, and it may be sudden in its attack, or be preceded by some days' illness, in which case the most prominent and characteristic symptoms in the adult are severe muscular pains simulating rheumatism, especially in the small of the back, and the frequent occurrence of obstinate vomiting, foreboding a severe form of the disease.

On the fourth day inclusive from the first attack of the primary fever, sometimes sooner, and but seldom later, the eruption appears, and the third stage commences. The phenomena of the third stage are as a calm succeeding to a storm; for, on the appearance of the eruption, the fever remits, the heat abates, the affection of the head subsides, the vomiting ceases, and the pulse returns to its natural standard, and consequently the febrile phenomena have altogether disappeared for the time. A temporary defervescence is thus well marked, the temperature falling, from perhaps 106° Fahr., progressively downwards to 100° Fahr.



TYPICAL RANGE OF TEMPERATURE IN A CASE OF NATURAL SMALL-POX COMMENCING WITH THE THIRD STAGE;  
 THAT IS, FROM THE PERIOD OF THE ERUPTION, ON THE EVENING OF THE FOURTH DAY FROM THE BEGIN-  
 NING OF THE SICKNESS. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wanderlich).

E. M. E. M. E. M. E. M. E. M. E. M. E. M. E. M. E. M. E. M. E. M. E. M. E. M. E. M. E.

LINE OF NORMAL TEMPERATURE, 98° FAHR.

The number of pustules varies, according to the severity of the case, from about twenty to some thousands. They appear first in minute bright-red specks on the face, neck, and upper extremities, then on the trunk, and lastly on the lower extremities, and run their course in a succession of crops. They undergo the various mutations of pimple, vesicle, and of pustule already described. About the eighth day of the disease, however, or when the eruption is fully out over the whole body, and the pustules on the face begin to mature, the whole face, head, and neck swell, particularly the eyelids, which often close and blind the patient; the swollen parts also throb, and are painful when touched. The intumescence of these parts lasts three days, during which the spaces between the pustules inflame, and are of a deep red or damask-rose color; and the closer this resemblance is seen to be, the milder will be the subsequent affections.

It is during this period of intumescence, simultaneously with the renewed hyperæmia of the skin, and introductory to the change taking place in the contents of the pustule, that the fever, which had remitted, returns, and the *fourth stage*, or that of secondary fever, commences—the *Fever of Suppuration*. This stage, in cases of ordinary intensity, is marked by a rise of temperature to a considerable height, by a frequent pulse, sometimes by a rigor, and by slight delirium, from which the patient is easily aroused. If, however, the disease be of greater intensity, hæmaturia, hæmoptysis, or a hard dry cough, are added. In favorable cases the swelling of the face, the redness of the intervening spaces, and also this secondary fever, having lasted from the eighth to the eleventh or twelfth day, subside, and the pustule, now fully ripe, bursts and discharges a thin yellow matter, which, concreting into a crust, falls off on the fourteenth or fifteenth day, and the disease terminates. During this somewhat protracted defervescence the temperature sinks gradually, to rise, perhaps, for the third time, when the desiccation takes place.

In the very mild variety of distinct small-pox which was wont to be named the "*horn-pox*," the primary fever is little more than a febricula; the pustules do not exceed half a dozen to two or three hundred; and, having passed through the stages of pimple and of vesicle, they, on the eighth day—*i. e.*, about the usual time of maturation—shrivel, desiccate, and crust. The secondary fever, often so fatal, does not recur, so that the convalescence usually commences on the eighth day, and the disease is terminated on the eleventh.

It was once supposed that in such cases the pus of the pustules was absorbed, but it appears that pus does not form, the fluid always remaining serous. In cases of any degree of severity, even in the *distinct small-pox*, the poison acts not only on the skin, but also on the buccal and conjunctival membranes, and produces an exudation on those parts. This additional affection, however, does not appear to aggravate the fever, or to occasion other inconvenience than what arises from the local disease. The buccal eruption is usually preceded and accompanied by soreness of the throat and difficulty of swallowing, and sometimes salivation; but these symptoms do not



Other symptoms, sometimes seen in the distinct small-pox, never fail to accompany the second stage of confluent small-pox—namely, *sore throat* and *salivation*. The tonsils and the fauces become tumid and red, the face begins to swell, and then the salivary discharge begins either with the eruption or within a day or two afterwards. The discharge of saliva is at first thin and copious, resembling the ptyalism of mercury. About the eighth day, however, it becomes viscid, and is expectorated with difficulty; while in bad cases it either ceases for a day or two, and then returns, or it disappears altogether abruptly; and if the swelling of the face also subsides suddenly, the danger is great. Children are not so liable to this salivation as adults. In them, however, a vicarious diarrhœa often appears, but not constantly; neither does it occur so early in the disease. It is frequently profuse, and often proceeds till the disease terminates. Not unfrequently the larynx and trachea are implicated, even to the larger divisions of the bronchia. There is cough, with hoarseness, painful expectoration, and sometimes complete extinction of the voice. These are most dangerous symptoms.

It has been stated that, on the appearance of the eruption and the commencement of the third stage, although the fever is mitigated, it does not altogether subside, defervescence is incomplete, and the affection of the head, the frequency of the pulse, and greater heat of the surface, often continue. With these ominous symptoms still present, on the eighth day of the eruption, or the eleventh day of the fever, the fourth stage, or secondary fever, commences, bringing with it new sources of anxiety to the physician and of danger to the patient. Gregory and Watson both consider the eighth day of the eruption as the most perilous day of the disease. Blood often appears in the urine in slight and sometimes in large amount. Renal cylinders are not uncommon. The bladder is affected in a great number of cases, and there is increased mucus. If the urine be retained in torpid and semi-comatose cases, it becomes soon ammoniacal, as in all cases with catarrhal cystitis (Parkes *On the Urine*, p. 262).

“The confluent small-pox,” says Sydenham, “does not in the least endanger life in the first days of the illness, unless there happens a flux of blood from the urinary passages, or from the lungs. Yet, on the decline of the disease, or on the eleventh, fourteenth, seventeenth, or twenty-first days, the patient is often brought to such a state that whether he will live or die is equally uncertain. He is first endangered on the eleventh day by a high fever (and the highness of the temperature may indicate the danger), attended with great restlessness, and other symptoms which ordinarily prove destructive, unless prevented by medicine. But should the patient outlive this day, the fourteenth and seventeenth are to be apprehended, for a very vehement fit of restlessness comes on every day towards evening, and there is the greatest difficulty in saving him.” The disease is apt to prove fatal by way of apnoea, after the eighth day; but after that period the characters of asthenia supervene.

The fatal symptoms of the fourth stage are, the absence of the usual redness in the intermediate spaces, the non-intumescence of



upon the eyes or near them ; none continued in bed an hour longer than they would have done in their best health."

The number of pustules is subject to great varieties, but, with very few exceptions, is much less than in the natural small-pox. In some cases not more than two or three appear ; occasionally only the primary pustule is seen ; but more generally the number varies from ten to two hundred, the mean being thirty or forty. Such is the general course of the inoculated small-pox. In a few instances, however, the disease that follows this operation is extremely severe, and in a still smaller number it is confluent ; and in either case the patient is perhaps destroyed. Many theories have been propounded to explain the singular mildness of the inoculated small-pox, but none of them are satisfactory.

**Complications of Small-Pox and Special Morbid Tendencies.**—Small-pox having been chiefly studied previous to any sound knowledge of morbid anatomy, or of morbid poisons, the occasional subsequent affections of the disease are still but imperfectly known. About the eighth day in the distinct small-pox, and the eleventh day in the confluent small-pox, a secondary fever is established, and at the same time a new series of phenomena may present themselves in a few severe cases,—as affections of the lungs, of the pleuræ, or of both ; of the urinary organs, or of the areolar tissue of the body generally. It is during the progress of this *secondary fever* that frequent opportunities occur for its degeneration into a fatal type. In such cases complete defervescence is never established ; but lesions become developed whose advent is capable of being appreciated by careful records of morning and evening temperature during the progress of this *fever of suppuration*. These are the *tertiary affections*, the eruptions and the fever being the *secondary effects* of the specific poison.

The most frequent affection of the lungs is hæmoptysis, but occasionally inflammation of these organs takes place, generally as pleuro-pneumonia. The mucous membrane, for instance, of the trachea is found often covered with a thick semi-purulent, muciform matter, peculiar to small-pox, irregular or honeycombed at its free surface, and which being removed, the subjacent tissue is found diffusely inflamed. The substance of the lungs also is occasionally found inflamed in every degree, even to purulent infiltration. The pleura also, according to Dr. Gregory, is peculiarly disposed to inflammation, which comes on about the eleventh or twelfth day, for the most part very suddenly, and proceeds rapidly to empyema, sometimes destroying the patient in thirty-six hours. The inflammation of the pleura does not merely run into suppuration, but takes every other form to which it is at any time liable.

The tertiary action of the variolous poison on the urinary and genital organs is seen in the frequent occurrence of hæmaturia, in the occasional formation of abscess of the kidney, in the occurrence of peripheric and parenchymatous orchitis, and in ovaritis ; while its action on the uterus is manifest from menorrhagia in the unimpregnated state, and by frequent miscarriage when the patient is parturient. The areolar tissue of the body generally is also acted



with his eyes." Further, he calculates that in 1000 cases 26 had ophthalmia, or about 1 in 39; and of these 11 lost an eye each, or 1 in about 100.

The inflammation of the buccal membrane may extend to the Eustachian tube, causing suppuration of the ear, and sometimes permanent deafness. It may spread also to the glottis; and the patient has been known to die suffocated by effusion into the areolar tissue around it, causing occlusion of the aperture. Sometimes it has terminated in ulceration, with the loss of a portion of the nose, or in caries of the jaw-bone, or in enlargement of the glands of the neck.

The soreness of the fauces and tonsils is often associated with pustules on these parts; and the tongue, the roof of the mouth, the inside of the cheeks, the uvula, and the *velum palati* may be covered with an eruption like pustules; and it has been much disputed whether the eruption forms on any other part of the mucous membrane. As a general principle, it does not; but Martinet found, in a man that died on the eighth day, the rectum covered with what he supposed to be variolous pustules. Rostan has seen the alimentary canal garnished with pustules similar to those of the mouth, from the œsophagus to the rectum. Sir Gilbert Blane also met with pustules on the mucous membrane of the intestines in two persons who died in the West Indies; and Rayer has given a plate representing pustules on the mucous membrane of the trachea. Dr. Mead's experience has made him state that, "I myself have seen subjects in which the lungs, brain, liver, and intestines were thickly beset with pustules." Dr. Pitzholdt, in the *Morbid Anatomy of Small-Pox*, writes that he has seen the peritoneum covering the liver and the spleen presenting appearances which he felt justified in regarding as the product of small-pox.

The pustules which form on the mucous membrane of the intestine, however, have not been very distinctly studied either as to their course or phenomena. Rayer terms them *rudimentary* pustules; and Dr. Watson believes the statement, that such pustules exist, to be a mistake.

A case of small-pox recorded by Dr. George Patterson, of Edinburgh, was examined by one of the most learned and discriminating pathologists of the day, Professor W. T. Gairdner. He observed pustules on the mucous membrane of the colon, and pronounced them to be identical with the pustules on the skin (*Edinburgh Monthly Journal*, 1849, p. 549). Still it appears to be doubtful whether such eruption on the mucous membrane of the intestine is not the same as that seen in cholera cases, extending (as I have frequently seen it do in cases I examined in the hospitals at Scutari, in 1855) throughout the whole intestinal tract. The appearance of eruption in such cases is due to the solitary mucous glands, which are filled with exudation, not of a purulent kind, but having all the external appearance of pustules.

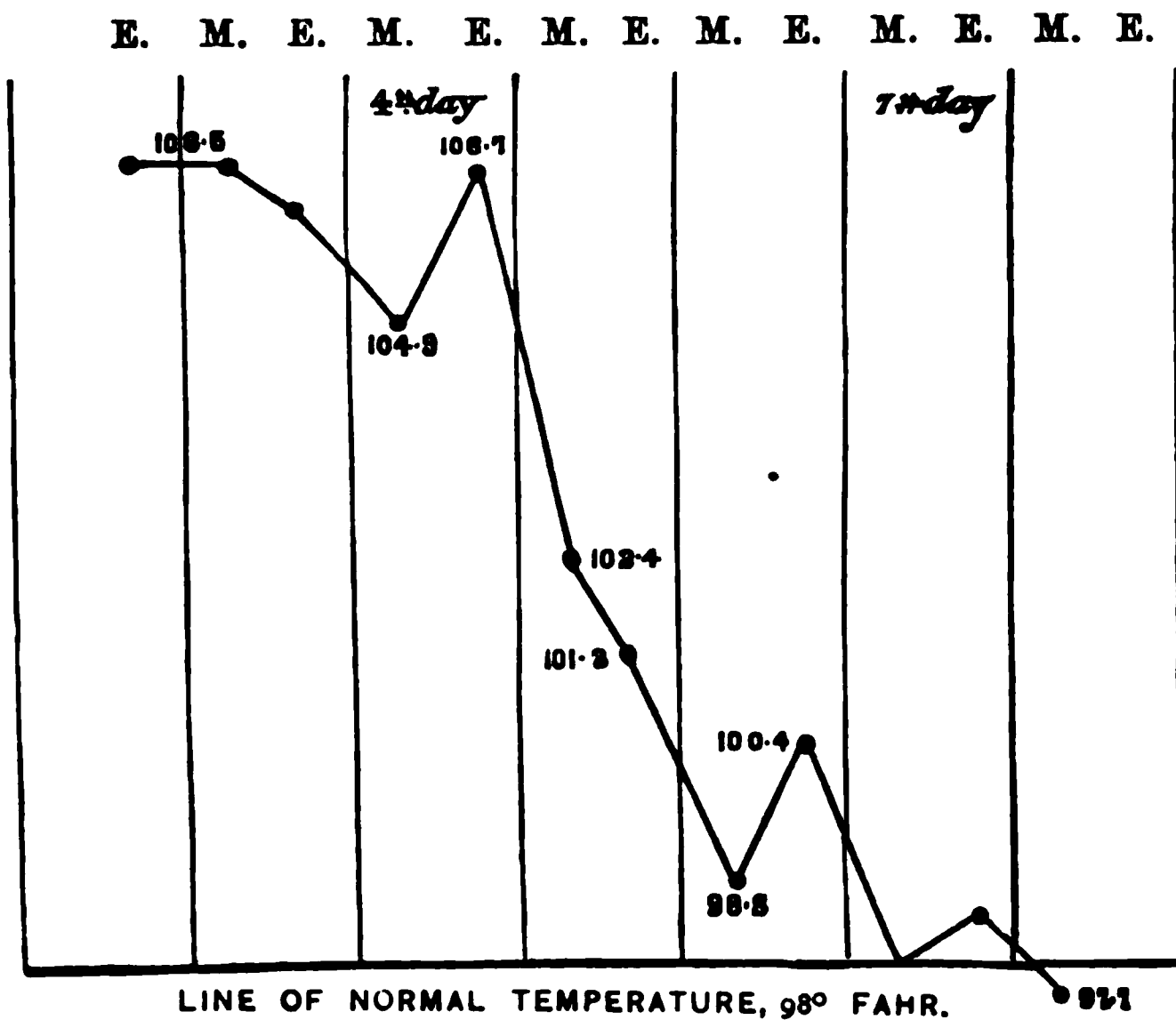
Such are the pathological phenomena which occasionally complicate small-pox. Death, however, not unfrequently anticipates their





thirty-six hours. From this event the patient remains entirely free from fever—*provided there exists no serious complication*—in spite of the continuous and progressive development of the small-pox pimples into pustules, and even in spite of the successive eruption of new pimples.

**TYPICAL RANGE OF TEMPERATURE IN A CASE OF SMALL-POX MODIFIED BY VACCINATION. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE SECOND DAY (Wunderlich).**



**Exhaustion of Susceptibility.**—The small-pox has the property, in common with measles and scarlet fever, of exhausting, on the first attack, the susceptibility of the constitution to the future actions of the poison. This law, however, is not without some exceptions, and in an epidemic at Marseilles, Bosquet considered that one person in one hundred was attacked a second time with small-pox. In some few instances even a second attack has no protective influence. Dr. Roupel says he met with an instance in which small-pox occurred three times in the same person. The lady of a Mr. Guinnett had it five times. Dr. Matson speaks of a lady who had it seven times; while Dr. Baron mentions a surgeon of the South Gloucestershire Militia who was so susceptible that he took small-pox every time he attended a patient laboring under that disease.

**Coexistence of Small-Pox with other Morbid States.**—The variolous poison is capable of *coexisting* with many other poisons, and also of influencing their actions, and of being reciprocally influenced by them. Dessessarz has seen variolæ coexist with scarlatina and with whooping-cough; Cruickshanks with measles; Frank with psora; and Dimsdale with syphilis. A patient was admitted into St.



pox—to be in the same room, or in the same house, with a patient laboring under the disease. It has been caught by passing a child ill of small-pox in the street; so that “to expose a person in the public highway, infected with this contagion, is considered a common nuisance, and indictable as such.” The dead body of a variolated person is equally infectious, and students who have been near it when brought into the dissecting-room have in consequence had the disease communicated to them, although they may not have touched the body (CÆSAR HAWKINS). The *infecting distance*, therefore, must be many yards around the patient’s person: indeed, with every precaution, there is great difficulty in preventing it spreading from ward to ward in large hospitals during the prevalence of the disease. “There is no contagion so strong and sure as that of small-pox: none that operates at so great a distance” (WATSON).

The fact that small-pox is communicable has been fully demonstrated by the once general practice of inoculation. The poison by this operation has been proved to exist in the serum, in the pus, and in the crusts of the small-pox pustule. There is no law more singular and unexpected, in the whole range of morbid poisons, than that the introduction of the variolous poison, by means of the cutaneous tissue, should produce an infinitely milder disease than when the same poison is absorbed by a mucous tissue. Then the poison seems to be much more uncontrollable in its operations, as in the case when it affects a person who breathes an infected atmosphere with one who has been *inoculated* with the small-pox poison inserted beneath his cuticle through a puncture of the skin. Several explanations are put forward, namely,—(1.) That the small quantity of the poison conveyed by inoculation into the blood may make the difference; (2.) That the disease is milder when the poison is admitted through the cutaneous than through the mucous tissues; (3.) It may be held that in passing through the absorbent mucous membrane the poison is not only admitted in large quantity, but its potency may be increased and its amount multiplied by the living cells of the mucous membrane through which it passes.

The causes which predispose to small-pox or increase the susceptibility of infection are,—(1.) A very early age. (2.) Not having had the disease before. (3.) Not having been vaccinated: such are called “unprotected persons.” (4.) Peculiarity of constitution—*e. g.*, the Negro and dark races. (5.) Fear of infection. (6.) Epidemic influence.

It is gratifying to know that of recent years the prevalence and mortality of small-pox in this country is greatly less than was wont to be. Dr. Farr tells us that, for the three years previous to 1855, out of every 1000 deaths from all causes, only 7.607 were from small-pox.

**Prognosis and Causes of Death.**—The prognosis of the natural small-pox is always most grave. The danger may be measured, to a certain degree, by,—(1.) The quantity and confluence of the eruption; (2.) The state of the circulating fluids; (3.) The presence and nature of the complications, especially those of the respiratory organs and



stage of continued fever. It is for the most part characterized by excitement rather than depression; and in the adult the muscular pains and pains in the back and loins are more severe and intense than in ordinary fever. The pain of the back is central in its position—a *spine-ache*—and is less affected by change of posture than the pain which is characteristic of lumbago, which affects the muscles at the side of the spine (often on one side only), and which is much aggravated by movement (BARCLAY). If vomiting occurs, which cannot be ascribed to any obvious cause, and persists till a papular eruption appears on the third or fourth day, with a remission of the febrile symptoms, little doubt can exist as to the variolous nature of the disease.

The diseases with which it may be, at first, confounded are, *petechial eruptions*, *measles*, and *chicken-pox*, and the *secondary pustular eruptions of syphilis*.

The nature of the fever, the character of the eruption, and the absence of any tendency to suppuration, are sufficient to distinguish *petechiæ* from variolous eruption.

Small-pox is to be distinguished from *measles* by the symptoms, as well as by the form and successive changes of the eruption. Crescentic patches, terminating in desquamation on the fourth day, characterize *measles*, as compared with small-pox, the eruption of which, even although it may be at first in efflorescent patches, never fails to become vesicular and pustular, proceeding to suppuration or blackening on the eighth day—a process which never fails to be attended by secondary fever.

It is more difficult to diagnose between varioloid and varicella, or chicken-pox. The chief difference consists in the eruption of chicken-pox presenting a vesicular character, which it retains; and it does not proceed to suppuration, but completes its course in five or six days, with a mild and short symptomatic fever.

The combination of mercury, scrofula, and syphilis often gives rise to cutaneous eruptions attended with fever, which may, in the first instance, be mistaken for small-pox. The eruption, however, is more tedious in its development, irregular in its course, and is persistent. It must, therefore, be distinguished by the history of the case, the long duration of the eruption, and the deep red or copper color it generally presents.

**Treatment.**—Since the first accounts by the Arabian physicians of the ravages of small-pox in Mecca, the history of this disease may be arranged in three great eras, each of which is characterized by remarkable epochs, and a fourth may be said to be now becoming apparent. The first of these eras is marked by an improvement in the treatment of small-pox. In few diseases has medical opinion undergone a more obviously beneficial change. To Sydenham is due the merit of this revolution in medical practice. The second era is marked by the discovery of the singular and beneficial phenomenon that the virulence of the poison of *small-pox* is greatly mitigated by introducing or ingrafting the disease into the system, through the cutaneous tissue, thereby causing the transference of the disease from one person to another, by inoculation. To Lady Mary Wortley



changed ; and, when the disease is long, the patient's back should be often examined, to prevent sloughing. The scalp likewise should be examined, and, if full of pustules, the hair should be cut off, to prevent its matting. If the disease be diagnosed early, however, it is proper to shave the scalp, because the irritation which attends the suppuration of the pustules is thereby diminished, and cold may be more efficiently applied to the head, if necessary. In the early stage of the primary fever, in severe cases more especially, it is necessary to have the bowels well opened in the first instance, and to keep them regular by saline medicine. A cathartic pill, composed of the following ingredients, will be found to be efficient in most cases, especially if aided by a seidlitz powder, given six or eight hours after the pill :

Two grains of calomel, one grain of the compound extract of colocynth, one grain of gamboge, and one grain and a half of scammony, made consistent with a little aromatic oil.

The bowels must be daily attended to afterwards, and castor oil, or rhubarb, or magnesia, &c., may sometimes be required. Saline diaphoretics, in the form of James's powder ; or the "aqua acetatis ammoniæ," to which a grain or two grains of tartar emetic has been added, so as to have  $\frac{1}{8}$ th or  $\frac{1}{16}$ th of a grain in every table-spoonful of the mixture, is an efficient and cooling diaphoretic. Spirit of nitric ether, or the nitrate of potass, may be added if required.

The surface of the body, over the hands, face, and feet, may be sponged several times a day with tepid water, with a view to relieve the intolerable itching ; but caution is necessary to prevent exposure to cold. Cold-cream, or a liniment of olive oil, glycerine, and lime-water, smeared from time to time over the itching surface by means of a camel-hair pencil, may be found to afford relief ; and chlorine lotions are highly spoken of by Eisenmann. With regard to the occurrence of convulsions in children, it is *not* found that opiates, as recommended by Sydenham and Cullen, are expedient. When the children are robust, or previously in good health, local bleedings, by means of one or two leeches to the temples, are more beneficial. Delirium, violent screaming, intolerance of light or sound, heat of head, all of which indicate a tendency to meningeal congestion, still more clearly warrant the application of leeches.

With regard to the propriety of bleeding (general) in adults, it is now well ascertained that it will neither eradicate the fever nor diminish the amount of the eruption. Bleeding is only warrantable if the pulse be full and strong, combined with evidence of inflammatory congestion in the lungs, liver, or brain.

When delirium, with restlessness, wakefulness, and a frequent pulse, is continuous, an opiate is indicated ; and, combined with tartar emetic, is most advantageously given. A draught composed of thirty minims of the solution of muriate of morphia, with half a grain of tartar emetic, will be found beneficial in such conditions, and especially when given at bedtime.

Cooling drinks of lemon-juice, tamarinds, neutral effervescing





mation can be rendered, so in proportion will the chance of "pitting" be diminished.

The local means adopted to prevent "pitting" may be shortly stated as follows ;

1. To open each individual pustule after suppuration has commenced.
2. To cauterize the pustule with nitrate of silver.
3. To employ both methods—that is, to open each of the pustules when it becomes vesicular, and introduce a strong solution of nitrate of silver into the cavity of the vesicle. At the end of a week scales fall off and no pit is left. Or lastly, to paint the face with a solution of nitrate of silver, in the proportion of one drachm of the nitrate to the ounce of water.
4. The application of a mercurial plaster, with the view of producing resolution of the *papulæ*. The preparation in use for this purpose at the Children's Hospital in Paris consists of 25 parts of mercurial ointment; 10 parts of yellow wax; 6 parts of black pitch.
5. Sulphur ointment applied several times a day.
6. Calamine mixed with olive oil, to form a coherent crust (BENNETT).
7. Tincture of iodine, painted over with a brush.
8. Saturated solution of gutta percha in chloroform (DRS. GRAVES and WALLACE).
9. To smear the face over with common olive oil.

All of these applications are for the most part applied to the face, the hands, and the arms only.

The severity and the mortality of small-pox has led many to think of means by which the disease might be completely extirpated. This leads us to consider—

## 2. *The Prophylactic, Sanitary, or Preventive Treatment of Small-Pox.*

Fifty years ago it was generally taught, among English physicians, that small-pox attacked the same individual only once in the course of life, and that its double occurrence in the same person was either very rare or next to impossible. The observations of Drs. Willan, John Thomson, Mr. Cross, Dr. Barnes, Dr. Craigie, and others since the time of these eminent physicians, lead to the following general conclusions:

1. Small-pox, though in general attacking the same individual only once during the course of life, may, however, affect him a second and even a third time.
2. This happens much more commonly when the first attack has been one of mild distinct small-pox than when it has been severe; and if the first attack has been one of confluent small-pox, it is rare for the same individual to have a second attack.
3. It is established by numerous observations, that an attack of any one of the varieties which have been named spurious small-pox or chicken-pox, by no means secures the same individual from an attack of confluent small-pox at a subsequent period.
4. Small-pox produced by inoculation does not necessarily secure the individual against an attack of small-pox induced in the natural way.



with an eruption and a fever, generally milder in form than small-pox acquired by breathing an atmosphere contaminated with the specific poison of the disease; and which thus passes through the mucous membrane to infect the blood. This is called the "natural way" of contracting small-pox; and the course of the disease so induced has been already noticed. For obvious reasons, the operation of *inoculating* the poison of small-pox has been rendered illegal in this country; and the practice of vaccination has been attempted to be enforced by law. What remains to be said about *inoculation* will be considered under the next topic.

### COW-POX.

LATIN, *Vaccinia*; FRENCH, *Vaccine*; GERMAN, *Kuhpocken*; ITALIAN, *Vaccinia*.

**Definition.**—*Cow-pox is the product of a specific and palpable morbid poison, which is reproduced and multiplied during the course of the malady in the cow or in the human being. After a definite period of incubation (from the time that the specific virus is artificially implanted, or communicated by impalpable emanations or effluvia in the "natural way"), specific pimples form upon some part of the skin, which pass through the stages of vesicle, pustule, scab, and desiccation. During the maturation of these specific pimples the adjoining lymphatic glands swell; a febrile state is induced, denoted by increase of temperature, constitutional disturbance of functions, acceleration of the pulse (which, to a certain extent, has been observed to continue persistent in some cases); and a general lichenous, roseolar, or vesicular eruption makes its appearance on the trunk of the limbs. The disease runs a definite course, affords immunity from another attack (for a considerable time at least), and exercises (during that period) a protective influence from human variola.*

**Pathology and Symptoms.**—The importance of a comprehensive knowledge of the pathology of variolous diseases generally, and of *cow-pox* in particular, lies in the relations of this latter disease to *small-pox* and to *vaccination*. Dr. Jenner named the disease "*variola vaccina*," implying thereby that one genus at least of the animal creation is liable to a disease of a kindred nature with that which attacks man. The disease in the cow was observed to be generally mild; in man it was observed to be most pestilential and fatal. It was observed, also, that the disease was communicable from the cow to man, and that persons so affected were protected from subsequent attacks alike of small-pox and of cow-pox. Dr. Jenner believed that the two diseases were in reality identical. It has now been shown by unquestionable evidence that cattle and horses have for centuries been known to be affected with a species of small-pox or variola. Every different writer who has seen the disease has given it a similar name. Previous to 1745 it was known and described in Italy (FRACASTORIUS, LANCISI, RAMAZINI) as a malignant disease which destroyed cattle almost as extensively as small-pox did the human race. It was first observed in this country in 1745, and again in 1770 it appeared among the horned cattle with so much severity that His



October, 1840. Two cottages, in which three persons resided during their illness, were situated on each side of a long narrow meadow, comprising scarcely two acres of pasture-land. One of these three patients, though thickly covered with pustules of small-pox, was not confined to her bed after the full development of the eruption; but frequently crossed the meadow to visit the other patients—a woman and child—the former of whom was in great danger, from the confluent malignant form of the disease, and died. According to custom, she was buried the same evening; but the intercourse between the cottages across the meadow was still continued. On the day following death the wearing apparel of the deceased, the bed-clothes and bedding of both patients, were exposed for purification on the hedges bounding the meadow; the chaff of the child's bed was thrown into the ditch; and the flock of the deceased woman's bed was strewed about on the grass over the meadow, where it was exposed and turned every night, and for several hours during the day. This purification of the clothes continued for eleven days. At that time eight milch cows and two young heifers (sturks) were turned into this meadow to graze. They entered it every morning for this purpose, and were driven from it every afternoon. Whenever the cows quitted the meadow the infected articles were again exposed on the hedges, and the flock of the bed was spread out on the grass, and repeatedly turned. These things remained till the morning, when the cows were re-admitted, and the contaminated articles were supposed to be withdrawn. It appears, however, that the removal of the infected articles was not always accomplished so punctually as had been enjoined, so that, on one occasion at least, the cows were seen in the midst of them, and licking up the flock of the bed which lay on the grass. These cows were in perfect health when first put out to graze in this meadow; but in twelve or fourteen days *five* (out of the eight) milch cows appeared to have heat and tenderness of the teats. The teats became swollen, and small hard pimples could be distinctly felt upon them, as if imbedded in the skin. These pimples daily increased in magnitude and tenderness; and in a week or ten days they rose into *blisters* (vesicles), passing into brown or blackish scabs. When the teats were in this condition, and very tender, constitutional symptoms of ill-health became developed. Sudden *sinking* or loss of milk, drivelling of saliva from the mouth, frequent inflation and retraction of the cheeks, staring of the coat, "tucking up of the limbs," "sticking up of the back," and rapid loss of flesh were the appearances which even the peasants themselves were able to appreciate. By the middle of the third week the pustules were mature, and the crust and loose cuticle began to be detached. The simultaneous occurrence of the disease on all the animals increases the probability of the operation of one common cause. The whole of the cows were certainly affected within less than three days of each other; and another circumstance requires particular notice, namely, the occurrence of the disease in a young heifer (stark), to which of course the disease could not have been communicated by those casualties which commonly propagate the vaccine variola amongst



communicated the vaccine disease from man back to the cow (*retro-vaccination*, as it has been called); and he has observed that good human lymph, when *re-transmitted* in this manner, loses some portion of its activity. The phenomena appear later, small vesicles are produced, but ultimately, *after successive re-inoculations on man*, it regains its activity. Human small-pox has also been transmitted through the horse to the cow, and so to the child in the form of cow-pox (FLETCHER).

As the first origin of these specific poisons is as yet unknown, it cannot be now definitively determined whether man first had the disease communicated to him from the animal creation, or whether the lower animals, such as horses or oxen, had the disease communicated to them from man. The existence of small-pox in man is recorded in China as early as 1122 years before Christ (MOORE). And it is certain that when variolous disease appears among the lower animals in a malignant form, it is capable of producing, by inoculation, a disease of similar severity in man, if he has not already suffered from a similar affection; and that the direct inoculation of the cow with human small-pox produces a mild and mitigated form of disease—that such disease being again reproduced in man by inoculation from the mitigated disease of the cow, accords entirely in its character, in its progress, and in its protecting influence with the *variola vaccinae*, as described by Dr. Jenner. These and similar facts seem to lead to the conclusion that small-pox and cow-pox are not dissimilar diseases, but are identical in their nature.

There are some remarkable circumstances which must at once arrest the attention of the student, who carefully studies the accounts given of the experiments on men and animals, from which many of these statements are deduced. *First*, There seems to have been great uncertainty and difficulty often attending the actual attempts to transfer the specific virus of these eruptive or variolous diseases from one animal to another. The very interesting experiments of Ceely, and of Thiele and others, demonstrate this in a remarkable manner. *Second*, These experiments show the *marked improvement which sometimes takes place in the energy, and therefore in the quality of the specific virus, by subsequent removes or inoculations, in animals of the same kind, after the virus had been successfully implanted in one of them*. This energy and improved quality was shown in the more perfect development of vesicles, and in the more active manifestations of the primary and secondary symptoms. The subsequent inoculations of such improved lymph seem to produce less severe and less dangerous *local* results—the virus seems less acrid, less virulent, and less mischievous—having apparently acquired increased specific activity combined with mildness of action, and a greater susceptibility of transmission from one animal to another of the same kind.

Keeping, therefore, these facts in view, the history of the remarkable epizootic of *variola orinae* which made its appearance in August, 1862, in some of the largest flocks of sheep in the West of England, is of great interest to the Pathologist. This variolous disease of





CARRO, RING). Fontan relates that some mares being affected with a pustular eruption, the matter from the pustules was inoculated on the teat of a cow, where it produced several fine pustules. From these several infants were vaccinated, with the result of producing perfectly characteristic vaccine vesicles. Thirty infants have been vaccinated from this source at Toulouse, and in all the result has been most satisfactory (*L'Union Méd.*, 1860; *New Syden. Society Year-Book*, 1860). If this can be definitely established, then the successful inoculation of some animal, *other than the sheep*, with the virus from the specific eruptive disease of the horse may give such energy and, at the same time, mildness to the morbid poison, by subsequent removes, that the implantation of the new virus (*equination*) may perhaps be followed by the same beneficial results to sheep, in respect of the malignant variolous disease to which they are liable, that *vaccination* has conferred on man in respect of small-pox. The question, then, at once suggests itself: "Has human small-pox ever been communicated to sheep, with the view of obtaining a modified lymph which may confer protection on them from the variolous disease to which they are liable?" From analogy, may we not indulge the hope that the practice of inoculating sheep from the small-pox of man might induce as mild and modified a disease in them, and prove as protective to them, as vaccine variola, through vaccination, has been to man? Or, having communicated the human variola to cows (as the experiments of Ceely and Thiele demonstrate that such may be effected), might not sheep be tried with the resulting virus as a protective agent? The vesicular eruptive diseases of dogs, as well as of horses, should be similarly inquired into and experimented with, seeing that dogs are so much associated with sheep.

The outbreaks of the variolous diseases amongst cattle and sheep seem to follow similar inexplicable paths to those which small-pox amongst human beings is observed to follow. Occasionally the disease is *epizootic* (equivalent to *epidemic* amongst men), or prevalent at the same time in several farms at no great distance. Cases spring up like small-pox, now and then, which appear to be solitary, and the source of which cannot be traced. It is rare indeed that the solitary cases of small-pox in human beings can be traced to a communicating source. In oxen it may be seen sometimes at contiguous farms; at other times, one or two farms, apparently similarly circumstanced amidst the prevailing disease, entirely escape its visitation. Sometimes it is introduced into a dairy by recently purchased cows. On the other hand it has been undoubtedly communicated to cows from the vesicular disease of the horse, through the hands of the common attendant on both animals. There can be no doubt, also, that the disease often exists, although it is not observed; for the disease being mild, and the tempers of the animals good, little notice is taken of tenderness in milking, and so the existence of disease escapes detection.

There are *spurious* forms of the disease, which it is very necessary to be able to distinguish.

In the true cow-pox there is very slight manifestation of fever or



observers (GENDRIN, CEELY, and others). The cow—like children and the young of other animals, particularly high-bred dogs—is subject to a purely vesicular eruption, which makes its appearance about the ninth or tenth day of the vaccine disease. The vesicles of this eruption, within twenty-four hours, contain a pellucid serous fluid, raising the epidermis. On the following day they become turbid, the cuticle collapses or bursts, and a thin, brittle, flimsy crust forms, and speedily falls off. Successive crops continue to form and desiccate for three or four weeks.

**Primary Vaccine Lymph.**—To procure *primary* liquid vaccine lymph direct from the cow, in a condition fit for use, is a task of no ordinary difficulty. *Primary* crusts should be sought for on the lower part of the udder and around the base of the teats; and during a search for these it is not improbable that smaller vesicles of later growth may be found to yield efficient lymph. The best lymph is to be obtained from perfect vesicles, before they begin to point. After this period it is less to be depended on, particularly if very abundant, thin, or discolored. Pointed vesicles, when broken by violence, are rarely to be relied on. Entire unpointed vesicles, or vesicles with central crusts, should be sought for on parts where they are least exposed to injury—namely, on the lower and naked parts of the udder and adjoining bases of the teats. It is impossible to exercise too much delicacy in the proceeding. The puncture to liberate the lymph should be made with a sharp lancet as *near the centre* of the vesicle as possible; and the epidermis may be gently raised to a moderate extent around the discolored or most depressed part. Slight pressure with the blade of the lancet, or between the thumb and finger, will enable the operator to charge a few points or capillary tubes with the slowly exuding lymph. Punctures at the elevated and indurated margin of the vesicle are utterly useless. They only give vent to blood. Vesicles on which the central crust has begun to form are the most productive, particularly if the crust be small and the margin of the vesicle be tender, hot, and tumid; and small superficial vesicles are often more yielding than contiguous larger vesicles, which are more deeply seated or confluent.

Useful substitutes for liquid lymph, capable of communicating the vaccine disease, are—(1.) Amorphous masses of concrete lymph, found upon or in close proximity to broken vesicles. They ought to be colorless, like crystals of white sugar-candy; or of a light amber hue, resembling fragments of barley-sugar. (2.) Central crusts, irregular, rough, and more or less conical; the more transparent and nearer a dark-brown hue the better. (3.) Vesicular crusts or desiccated vesicles. These crusts should be carefully removed by the milkers before they are casually removed or spontaneously fall; and those only of primary formation, which are as it were the mould of a vesicle, of a dark-brown translucent appearance, should be retained. These three dry conditions of the specific vaccine virus may be reduced to a liquid state at any time for use. Glycerine is said to be the best solvent for such solid conditions of the lymph, which ought to be reduced to powder before the glycerine is added (Collins, *Boston Med. and Surg. Journal*, 1858).



is mainly given from a notice of the subject, written by the author, in the pages of the *Medico-Chirurgical Review* for 1857:

In 1841 the Vaccination Act was passed, which rightly made the practice of *inoculation unlawful*. In 1853 another Act was passed, with the view of rendering the practice of *vaccination compulsory*,—an Act which is known as Lord Lyttleton's Vaccination Act. During the interval between the first and second reading of the Bill in the House of Commons, "The Small-Pox and Vaccination Committee of the Epidemiological Society completed a report on the prevalence and mortality of small-pox, and of the means taken to guard against it through vaccination." The conclusions they arrived at were deduced from the largest and most accurate mass of statistical evidence which had ever been brought to bear upon the question, and were eminently calculated to encourage Her Majesty's Ministers to pass an efficient measure to *compel* vaccination. A most valuable pamphlet was afterwards published by Dr. Seaton, which demonstrates the truth in a still more forcible manner, as to the protecting and modifying influence of vaccination in small-pox. To this belief, indeed, the general assent of the medical profession appears to have been given at least fifty years ago. *Then*, it would seem to have been all but unanimous; and *now*, one would think, at first sight, that it were almost an insult to human understanding to be obliged to collect statistics to *prove* that vaccination confers a large exemption from attacks of small-pox, and almost absolute security against death from that disease. But so it is, and independently of the information which such statistical inquiry is calculated to convey to those who advise our Lawgivers and Public Administrators, the inquiry is eminently useful in relation to everything which bears on the nature of vaccine and variolous disease. The general ignorance of the community, especially of the lower orders, as to the aim and object of vaccination, is lamentably great, and has still to be overcome. Moreover, the highest medical authorities of late years recommend that all views and facts put forward as objections to vaccination should be vigorously inquired into, and that there should be published from time to time a true account of such inquiries, with an elucidation of what has seemed doubtful and contradictory (SIGMUND, ALISON).

It is now well known that Lord Lyttleton's Vaccination Act (1853) has proved but a very imperfect measure—a piece of legislation which has fallen very far short of accomplishing all that is yet required. The inefficiency and imperfect working of the Act has been fully shown,—(1.) In the Reports of the Registrar-General for 1854; (2.) By the medical profession generally; (3.) By the medical registrars in particular; (4.) By the public, as expressed now and again in the newspapers of the day. To this state of things we owe a most valuable work on vaccination, written by the indefatigable medical officer (JOHN SIMON, F.R.S.) of the then (1857) General Board of Health. The aim of this publication was to lay before the Board such medical facts and considerations as might assist in estimating the hygienic value of vaccination, and the strength of any objections which may have been alleged against its general adoption.



by comparing the statistics of vaccination from various German States with similar statistics from different districts in Great Britain and Ireland, that where vaccination is most perfectly carried out, small-pox is least mortal. The following are the general results which the Committee of the Epidemiological Society arrived at:

1. To prove the influence of vaccination in England, it is shown that out of every 1000 deaths in the half-century from 1750 to 1800 there were **96** deaths from small-pox; and out of every 1000 deaths in the half-century from 1800 to 1850 there were only **35** deaths from small-pox.

2. To prove the influence of vaccination on the Continent, it is shown that in various German States sufficient evidence can be obtained to show that out of every 1000 deaths *before vaccination was used*, **66.5** were deaths from small-pox; but that out of every 1000 deaths *after vaccination* came into use, the deaths from small-pox were only **7.26**.

3. To prove that in countries where vaccination is most perfectly carried out small-pox is least mortal, it is shown that—

(a.) In this country, where vaccination has been voluntary, and frequently neglected, the deaths from all causes being 1000, the deaths from small-pox were as follows:

London, . . . . .	16	Edinburgh, . . . . .	19.4
Birmingham, . . . . .	16.6	Glasgow,* . . . . .	86
Leeds, . . . . .	17.5	Galway,* . . . . .	85
England and Wales, . . . . .	21.9	Limerick,* . . . . .	41
Perth, . . . . .	25	Dublin, . . . . .	25.6
Paisley, . . . . .	18	Connaught,* . . . . .	60
All Ireland, . . . . .			49

(b.) In other countries, where vaccination has been more or less compulsory, the deaths from all causes being 1000, the deaths from small-pox were as follows:

Westphalia, . . . . .	6	Bohemia, . . . . .	2
Saxony, . . . . .	8.88	Lombardy, . . . . .	2
Rhenish Provinces, . . . . .	8.7	Venice, . . . . .	2.2
Pomerania, . . . . .	5.25	Sweden, . . . . .	2.7
Lower Austria, . . . . .	6	Bavaria, . . . . .	4

Evidence corroborative of these results has been adduced by Dr. Balfour from the records of the Army and Navy Medical Departments, where every soldier or sailor is protected by vaccination, if he has not previously suffered from *cow-pox* or *small-pox*.

1. For twenty years, namely, from 1817 to 1837 inclusive, it is shown that in Dragoon Regiments and Guards, with an aggregate

---

\* With regard to the high rate of small-pox mortality in the towns marked by the asterisks, it was clearly shown by Dr. Stark, in Edinburgh, and by Dr. J. C. Steele (the present Medical Superintendent of Guy's Hospital in London), that such mortality was due to the neglect of vaccination. Dr. Steele, then resident in Glasgow Infirmary, called attention to the great increase of small-pox in Glasgow, as mainly coming from the Highland and Irish population, among whom vaccination was rare. Dr. Stark showed that more than eighty per cent of all the deaths from small-pox happened in children under five years of age.





2. During sixty-three years in which *vaccination* was practised, and that to a very great extent, there was 53 distinct and well-marked epidemics; which is equal to a ratio of **84** epidemics in 100 years.

3. During the fifty-five years since *vaccination* has been mainly practised, there have been 12 distinct and well-marked epidemics of small-pox; which is equal to a ratio of **24** epidemics in 100 years.

This kind of testimony is greatly enhanced by the fact, that epidemics never occur in the army or navy of our own country, nor in those countries where the soldiers and seamen are efficiently protected by vaccination. The details given with reference to the two Malta epidemics in 1830 and 1838 afford a striking proof of the protective power of vaccination when tested by epidemic influence; and there are records of the Danish army and navy having altogether escaped during several epidemics of small-pox in Denmark.

There is still another way in which the protective power of vaccination makes itself manifest—namely, by *the mildness of the disease in the vaccinated compared with the unvaccinated, and the almost absolute security against death from small-pox which PERFECT vaccination confers*. With few exceptions, this appears to be the universal belief of the medical profession. At various times the opinions of large numbers of medical men have been specially asked for and obtained on this point. *Three* distinct and very comprehensive “*polls*” may be referred to, namely,—(1.) That by the College of Physicians in London, eight years after vaccination had been adopted. (2.) An almost national “*poll*” taken by the Epidemiological Society of London about the years 1852 and 1853. The written opinions of nearly two thousand medical men in this kingdom, as well as Bombay, Bengal, the Mauritius, the West Indies, and various other places, were here expressed; and they concurred in confirming the belief in the protecting and modifying influence of vaccination in small-pox. (3.) A very extensive “*poll*,” of which a list is published by Mr. Simon, comprehends not only members of the medical profession generally, but also the members of the Medical Department of the Army and Navy, together with the opinions of foreign governments.

These “*polls*” may be held as completely decisive of the question, really practically decided in the affirmative fifty years ago. From such evidence the inference is so inevitable, “that he who disputes it is equally unreasonable as he who opposes in like manner any proposition in Euclid” (ALISON).

The actual *extent of the security against death from small-pox* enjoyed by vaccinated compared with unvaccinated persons, has been calculated by Mr. Simon from various sources; and it appears that the death-rate from small-pox amongst the vaccinated varies from an inappreciably small mortality to  $12\frac{1}{2}$  per cent.; that amongst the unprotected the death-rate from small-pox ranges from  $14\frac{1}{2}$  to  $53\frac{1}{4}$  per cent.

The average percentage mortality from small-pox, stated by Mr. MARSH to occur amongst the vaccinated, is 5.24; but when vaccination is known to have been perfectly performed, as *shown by the*



to do. There is therefore the greatest necessity for vigilance on the part of every intelligent member of the community to prevent any re-accumulation of unvaccinated persons.

Four conditions are absolutely necessary to be efficiently carried out before we can hope to see small-pox eradicated through vaccination, and the aim of Jenner accomplished. These are,—(1.) The vaccination of every child must be made compulsory within a certain time after birth. (2.) *Systematic inspections* of two kinds must be constantly and periodically made by competent persons—namely, one to ascertain as to the effectual performance of the operation, as evinced by the kind of cicatrix visible. This inspection may be most conveniently made in public and private schools. Another inspection should have for its object to ascertain the numbers vaccinated within a certain territory compared with the numbers born in the same place. (3.) Every attempt at *variolous inoculation* ought to be made a penal offence. (4.) Every case of small-pox ought to be treated in strict seclusion, and be as completely as possible isolated, following out all the directions given with reference to the management of epidemics at page 226, as are applicable to the case. A quarantine regulation to enforce segregation of the sick from small-pox is of far more importance in this country than for yellow fever, which does not find a *habitat* with us. We come now to consider—

II. *How the Protective Influence of Vaccination has been Impaired.*—Since vaccination has been generally practised it has now and then seemed apparent that “*the protective power of vaccination becomes gradually weaker, and at length dies out in the individual.*” The works and reports which have been mentioned seem to demonstrate the truth of this statement; but in justice to Dr. Seaton (one of the greatest authorities on the subject) it must be stated that he does not subscribe to this belief. Indeed, in his last Report (*Appendix to Public Health Report* for 1861, p. 64), he states that where uniform care in the selection of lymph, and in the performance of the operation, was practised, the results did not favor the hypothesis that there had been any necessary deterioration of the lymph. He has seen several cicatrices, the results of the vaccinations of Dr. Jenner and Dr. Walker; but the work of the vaccinators to whom he refers (and mentions as having bestowed great care in the selection of their lymph and in the performance of the operation) will bear comparison with the results obtained by Dr. Jenner and Dr. Walker.

In 1809 Mr. Brown, of Musselburgh, near Edinburgh, published the opinion that the prophylactic virtue of cow-pox diminished as the time from vaccination increased. In 1818 and 1819 small-pox prevailed in Scotland as an epidemic, and many vaccinated persons passed through a mild form of variola. The terms “modified small-pox” and “varioid disease” about this time came into general use; and two classic monographs on the subject made their appearance, one by Dr. Monro, in 1818, and another by Dr. John Thomson, of Edinburgh, in 1820. Dr. Copland also writes that he saw and described, as early as 1823, small-pox as it affected members of the same family at different periods after vaccination, and in young persons who had been vaccinated only ten or eleven years. Contrasting such



issued by circular, of date 21st September, 1858, and is at present in force, which ordains that "every recruit, without exception, on joining the Head-Quarters or Depot of the Corps or Regiment to which he belongs, shall be vaccinated, even if he should be found to have marks of small-pox or of previous vaccination, and that a monthly return of the results (as to (1) a perfect vaccine pustule following the operation, or (2) a modified one, or (3) a failure) shall be forwarded to the Director-General" (*Statistical Report for 1859*, p. 21).

On the other hand, it must be remembered, as Mr. Marson clearly shows, that "probably *re-vaccination* does not afford the same amount of protection that the first vaccination *well performed* does. The great object to aim at is to vaccinate *well* in infancy. This should be looked upon as the sheet anchor; and therefore a careless vaccination should be deprecated at all times, practised under the belief that, if it fails to take effect properly, it will be of no consequence, as the operation can be repeated. By such a proceeding the vaccination often takes effect *badly*, and will never afterwards take effect *properly*, and the individual may take small-pox *severely*."

It has been alleged (but sufficient proof has not yet been adduced to show) that the vaccine virus becomes deteriorated by its passage through numerous human bodies. In other words, it has been supposed that its protective influence is weakened by length of time or of use, in consequence of the long succession of subjects through whom it has been transmitted since its direct inoculation from the cow. This doctrine is opposed to the obvious pathological fact, that the specific virus of cow-pox, small-pox, and other similar diseases, multiplies and reproduces itself in the system of those who suffer in the natural course of these diseases. Considerable differences of theoretical opinion prevail upon the point. In the report of the National Vaccine Establishment for 1854 it is stated "that the vaccine lymph does not lose any of its prophylactic power by a continued transit through successive subjects." Such an unqualified belief is not, however, by any means universal, as shown in various parts of the evidence collected by Mr. Simon. It is certain that the vaccine lymph, when taken direct from the cow, seems to show an amount of infective power which is not usual in lymph of long descent; but how much of this effect is due to irritation simply, and how much to specific action, does not seem certain. Lymph direct from the cow "takes" (as the phrase is) in persons with whom lymph of long descent has failed. This is more often obvious in re-vaccinations. Lymph direct from the cow excites local changes of an intenser kind, so active, indeed, as to render caution necessary in its selection and use. The vesicle produced by it runs a full course, compared with which the progress of vaccine vesicles from lymph of long descent seems unduly rapid, and their termination premature. Also, the lymph direct from the cow renders more certain, and apparently more characteristic, the slight febrile disturbance which is proper to the action of cow-pox on the human system. This febrile disturbance is undoubtedly an essential pathological phenomenon, demanded alike for the due protection of the vaccinated



ness in taking lymph at its perfection, and by neglecting to observe the rule never to use lymph or crusts not perfect in all respects, and free from blood or pus, frequent instances of inoculation with purulent matter or unhealthy blood happened.

(4.) By the use of matter, fluid or concrete (purulent or morbid in either case), taken from sores of any specific and enthetic character, as (a) erysipelatous and ecthymatous; (b) that of zymotic ulceration and destruction of tissues, and possessing the properties of a morbid poison; (c) syphilis, primary or secondary, whether communicated by a lancet, or contaminated vaccine points or crusts; or, as occasionally happened, the manifestation of syphilitic phenomena in connection with, or supervening upon, genuine or spurious vaccination.

(5.) The deterioration of genuine virus, by transmission through scorbutic and unhealthy persons, or where at the time of re-vaccination the protective power of a former vaccination was partially retained, or to the continued use of virus from adult soldiers many of whom were suffering from unhealthy influences, instead of using lymph from the primary vesicles of healthy infants.

(6.) The destruction or deterioration of originally good virus by heat and humidity.

These results substantiate the observations of Jenner as to the necessity of guarding against deterioration of the virus of cow-pox, which losing its specific property, ceases to be prophylactic. Army experience would go to prove that genuine vaccination is an absolute safeguard against small-pox (DR. ELISHA HARRIS, *Contributions relating to the Causation and Prevention of Disease, &c.* Published by the United States Sanitary Commission, 1867).]

**The Operation of Vaccination** ought to be performed in childhood, and it is ordained by law in this country to be performed within *three*, or in case of orphanage within *four months* of birth. The infant ought to be, at least, from *four to six weeks* old, before a disease, sometimes attended with considerable febrile disturbance, is ingrafted upon the constitution. Under *six weeks* of age, infants should never be vaccinated, unless in cases of urgent necessity, such as small-pox being in the vicinity. The age of *three months* is on the whole to be preferred. The child ought to be in good health, free from any eruptive cutaneous disease, and free from disorders of teething, of the bowels, or other diseases peculiar to the age of childhood, otherwise the protective influence of the vaccination cannot be depended on.

Difference of opinion exists as to the number of vesicles it is proper to graft upon the arm, and the size of them. Some believe the person to be as thoroughly protected by a small vesicle, "the tenth of an inch in diameter, as if the arm were covered with inoculated points" (CAZENAVE, ANDREW ANDERSON); and many vaccinators regard the multiplication of vesicles only as a safeguard against failure, and attach value to *one* successful insertion only of the vaccine lymph (Buchanan, *Appendix to Fourth Report on Public Health*, for 1861, p. 111). On the other hand, the official instructions issued to vaccinators in England contain the following directions: "In all ordinary vaccinations, vaccinate by *four or five separate punctures*, so as to produce *four or five separate good-sized* vesi-





3. Simple abrasion of the cuticle is sometimes resorted to with very good success—namely, by scraping off the cuticle with the lancet, used as an eraser is used to remove blots from paper (*Fourth Report on Public Health*, p. 107).

**Signs of Successful Vaccination.**—By the end of the *second* day small spots appear elevated over the sites of the punctures, or over the groups of scratches or abrasions; and these, when examined by a simple lens, are seen to be vesicular, and surrounded by a slight redness. This stage continues for three to four days from the date of ingrafting the virus. About the *third*, but rather towards the *fourth* day, the elevation is more perceptible and more red; and by the *fifth* or *sixth* day a distinct vesicle is obvious upon it, of a whitish color, having a round or oval form, an elevated edge, and a depressed centre. Late on the *seventh*, or early on the *eighth* day, an inflamed ring or areola begins to form round the base of the vesicle, and with it continues to increase during the two following days. This areola is of a circular form, and its diameter extends from one to three inches. *On the eighth day the vesicle appears distended with a clear lymph. This is the day of its greatest perfection, and it is the proper period for obtaining the specific virus for continuing vaccination on others. The vesicle is now circular and pearl-colored; its margin is turgid, firm, shining, and wheel-shaped.*

Having reached its height on the *ninth* or *tenth* day, the development of the bright-red areola is accompanied with considerable tumefaction of the skin, with hardness and swelling of the subjacent areolar tissue. This erythematous ring is often the seat of small vesicles. By the *tenth* day, also, the febrile symptoms of constitutional disturbance are well expressed, the lymphatics of the arm are engorged, and sometimes a roseolous rash supervenes over the body. On the *tenth* or *eleventh* day the areola begins to subside, leaving, as it fades, two or three concentric circles of redness. The vesicle now begins to dry in the centre, and acquires there a brownish color. The lymph which remains becomes opaque and gradually concretes, desiccation commences, and tumefaction subsides, so that by the *fourteenth* or *fifteenth* day the vesicle is converted into a hard round scab of a reddish-brown color. This scab contracts, dries, and blackens, and about the *twenty-first* to the *twenty-fifth* day from the date of vaccination may fall off. It leaves a cicatrix which commonly is permanent in after-life. Indeed, the mark of a good cicatrix is indelible if it is not injured (GREGORY, MARSON, CEELY, CAZENAVE, SIMON).

While these local changes are in active progress, febrile phenomena become established—first, so slightly from the *fifth* to the *seventh* day, that often the fact passes unobserved; and again, more considerably during those days when the areola is about its height. The patient is then restless and hot, with more or less disturbance of stomach and bowels. About the same time, especially if the weather be hot, children of full habit not unfrequently show on the extremities, and less copiously on the trunk, a lichenous, roseolar, or vesicular eruption, which commonly continues for about a week. When vaccination is performed on such adults or



operation. Scars also having a less diameter than *a quarter* of an inch ought to find a place amongst this class; and generally, all ill-defined, faint, scarcely discernible white patches, especially such as consist of large, flat, ill-defined shiny marks. Fruitless attempts at vaccination may be also recognized by the permanent traces left of the parallel or transverse scratches employed at the operation.

It is, however, very difficult to describe the extent of differences between the results produced by different vaccinators. A large amount of *bad*, and a still larger amount of *second-rate* vaccination has been found to prevail in many districts, as the result of the inspections instituted by Mr. Simon in 1860 and 1861 abundantly testify. Medical men are found to vary exceedingly in their estimate of a satisfactory vaccine vesicle and cicatrix, or the reverse, for the standard is comparative rather than absolute (SEATON, SANDERSON, BUCHANAN). This is exactly what might have been expected, seeing that medical students are left to pick up their knowledge of vaccination where they can. In fact, practical medical education at our schools of medicine has hitherto, or until very recently, been entirely *nil* in regard to this most important subject; and no test of knowledge has ever been applied. Many men, whose estimate of the quality of the resulting cicatrices is of a low standard, can scarcely appreciate the typical character of marks which are the ordinary results of good vaccination (SEATON). Excessively small cicatrices are apt not to be perfect, and there are great varieties in the size of cicatrices of perfect character, the results of puncture. It is therefore fairly presumed that cicatrices which thus vary cannot all have precisely the same value. The hand of different vaccinators can even be recognized by the kind of marks they leave behind them. The marks of some vaccinators are conspicuous for their excellence; the marks left by others are not so; and hence there are great differences between the vaccination of districts where different vaccinators are employed. In the schools, for instance, of large towns, Mr. Seaton informs us that "where the work of many vaccinators was seen together, it was frequently possible to fit the work to the vaccinator by the kind of cicatrix."

With regard to the means of estimating the efficiency of vaccination, it seems established that "*a distinct connection subsists between the NUMBER and the QUALITY of the cicatrices and the protection conferred by vaccination against small-pox; so that it may be confidently stated that that vaccination is the most efficient from which the most and the best cicatrices result.*" The evidence derived from the records of the Small-Pox Hospital, collected by Mr. Marson, regarding the superior value of several rather than few vesicles, appears to be conclusive on this point.

These facts have been tabulated by Mr. Simon in the following form, as the result of observations made during twenty-five years, in nearly 6000 cases of small-pox contracted after vaccination, the persons having been vaccinated in different ways as regards the *number and quality* of the cicatrices:



of glass glued together by the dried lymph, or on lancets. These should be well charged—i. e., coated twice, or even thrice with the lymph, and rolled up in a covering of goldbeater's skin, and still further secured from atmospheric influences by an outer case of tin-foil hermetically sealed, or in a vial carefully corked, in which they may be packed with cotton, if they require to be transmitted to any distance. Glycerine has been used with success to keep the lymph liquid.

By proper care, complete and perfect vaccination may be attained under every variety of method; but bad vaccination, as it prevails at present, is almost always directly dependent on the careless employment of improperly preserved *dry* lymph, and indirectly associated with irregularity of inspection, in consequence of which the vaccinator remains unaware of the number and extent of his *failures*, and loses all the advantages of experience. "The use of the capillary tubes of Dr. Husband affords considerable advantages to the public vaccinator, especially if his district be rural—*Firstly*, Because it furnishes him with an efficient means of maintaining his supply without having recourse to extraneous sources, and thus enables him to dispense altogether with the use of 'points,' 'glasses,' &c.; *Secondly*, Because in thinly populated neighborhoods, in which experience shows that it is impossible to assemble all the children at any particular station, it enables him with equal advantage to vaccinate from house to house" (Sanderson in *Public Health Report*, 1861). For a detailed account of Dr. Husband's method of preserving lymph, the reader is referred to the *Second Report of the Medical Officer of the Privy Council*, 1859.

### CHICKEN-POX.

**LATIN**, *Varicella*; **FRENCH**, *Varicelle*; **GERMAN**, *Windpocken*—Syn., *Wasserpocken*; *Varicellen*; **ITALIAN**, *Varicella*.

**Definition.**—*The disease consists of a specific eruption, in a series of crops, on the breast, back, face, and extremities, attended with fever, which runs a definite course in eight or ten days.*

**Pathology.**—This disease derives an importance which it does not of itself possess, in consequence of its resemblance to small-pox, with the modified form of which it is considered by some to be identical. It is for the most part peculiar to childhood and early adult age; but its epidemic influence is very inconsiderable, and its extension easily under control. That it is communicable has been proved by inoculation. The theory of the disease, therefore, is, that a specific poison, after a given period of latency, gives rise to primary fever which lasts from twenty-four to seventy-two hours, when the eruption appears, and runs a course of eight or ten days. The fever is much mitigated on the appearance of the eruption, and entirely subsides with it.

That fever precedes the eruption is a phenomenon observed so generally that no exception is to be found in the account given of



ments (CRAIGIE) embrace the most important pathognomonic characters derived from the respective phenomena of both diseases:

1. Chicken-pox emits a peculiar odor, different from that of small-pox, and less decidedly partaking of the variolous fœtor.

2. Chicken-pox appears indiscriminately, and almost equally all over the person, beginning first on the trunk in general, and then appearing on the face and scalp; while small-pox appears first on the face and neck, and the pimples are more numerous on the face than on any other part.

3. Chicken-pox eruption is generally completed in the space of twenty-four hours, or solitary vesicles come out irregularly afterwards in different points; but in small-pox the eruption begins in the evening of the third, or morning of the fourth day, and proceeds regularly for the ensuing three days, until it is completely established.

4. While variolous pustules are on the first and second days of the eruption small, hard, globular, red, and painful, and communicate to the finger a sensation similar to that which would be excited by the presence of small round seeds under the cuticle; in chicken-pox, every vesicle almost has on the first day a hard red margin, but communicates to the finger a sensation like that from a rounded seed flattened by pressure.

5. On the second or third day of the eruption of chicken-pox, the individual bodies are vesicles containing serous fluid, and giving them a whitish aspect.

6. These vesicles are surrounded by little or no inflammatory redness, and do not naturally, and independent of external violence, proceed to suppuration.

7. Chicken-pox may be confidently distinguished from small-pox on the third and fourth days by the state of the vesicles, some of which, being left entire, are shrivelled and wrinkled, while others, whose ruptured tops have been closed by incrustation of their fluid, are marked by radiating furrows. None present depressions on the *apices*; and as they do not suppurate, they incrust and disappear sooner than variolous pustules.

8. The marks left by chicken-pox, when they do leave marks, present a peculiar conformation, being round or elliptical, and less frequently irregular than those of small-pox, and in general smooth and shining. Lastly, it is said by Luders, that while small-pox is formed in the *cutis vera* or corion, the chicken-pox eruption is formed in the tissue situate between the corion and cuticle (CRAIGIE, vol. i, p. 614).

**Treatment.**—It consists simply in abstinence from animal food, having recourse to a milk diet, and careful attention to the bowels. The patient is to be kept cool, by light coverings, and by making him repose on a mattress rather than on a feather bed.

## MILIARY FEVER.

LATIN, FRENCH, GERMAN, AND ITALIAN, *Miliaria*.

**Definition.**—*A disease in which there is an eruption of innumerable minute pimples, with white summits, occurring in successive crops upon the skin of the trunk and extremities, preceded and accompanied with fever, anxietas, oppression of respiration, copious sweats of a rank, sour, fetid odor, peculiar to the disease. The base of the pimples and the skin around are red and irritable.*





cated by nausea and vomiting of bilious matter, acid eructations, flatulence, and diarrhoea, frequently complicate the disease. Two forms have been described,—namely, a mild and malignant. The malignant is rendered so chiefly by the occurrence of violent inflammation in some of the internal organs, especially of the stomach, lungs, kidneys, or brain; and the danger of the disease is chiefly due to these complications. Such malignant forms have been known to prove fatal in two or three days, but more frequently in from seven to twenty-one.

The Treatment of the disease appears to consist in cooling drinks, purgatives, and antiphlogistics, as prescribed by the Italian medical officers who commonly attend on the sick in Turkey.

### MEASLES.

LATIN, *Morbilli*; FRENCH, *Rougeole*; GERMAN, *Masern*; ITALIAN, *Rosolia*.

**Definition.**—*The eruption in crops of a crimson rash, consisting of slightly elevated minute dots, disposed in irregular circular forms, or crescents; preceded by catarrhal symptoms for about four days, and accompanied with fever. It affects the system only once; and sometimes prevails as an epidemic. The eruption lasts six or seven days, and the whole duration of the disease is completed in from nine to twelve days.*

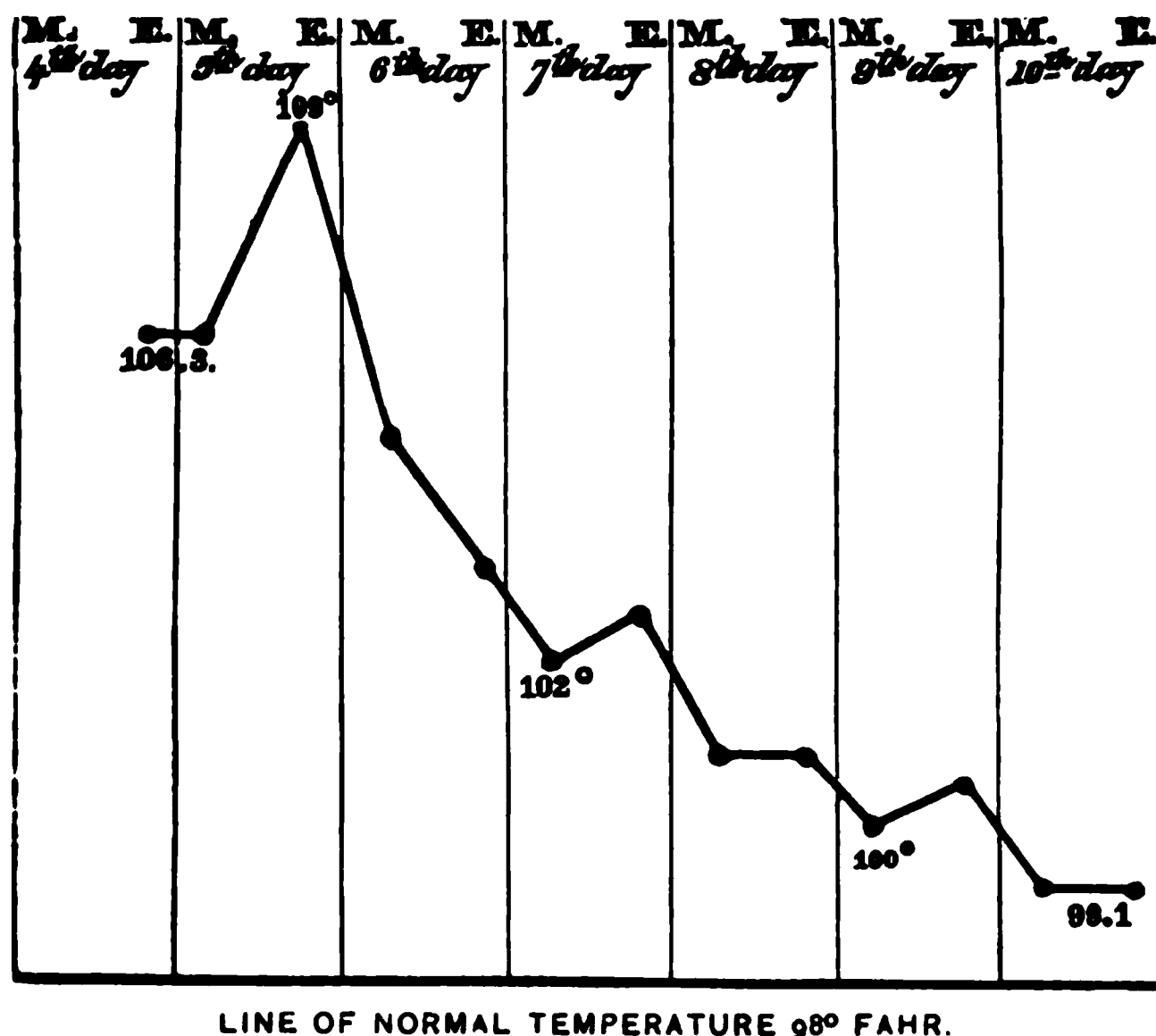
**Pathology.**—That a poison is absorbed in cases of measles, and infects the blood, there can be no doubt, inducing, after a period of incubation of thirteen or fourteen days, a continued fever, which does not remit on the appearance of the eruption. The fever thus established at the end of three, more generally of four, and in some few instances of five days, is followed by a certain secondary or specific inflammation of the skin and of the mucous membranes of the eyes, nose, mouth, fauces, and bronchia. In a few cases the poison has certain tertiary actions, and produces inflammation of the substance of the lungs, or of the pleura, which may greatly prolong the illness.

The pyrexia may greatly vary in intensity, but it is uniformly present. The fever which precedes the local lesions is termed the primary fever; and the premonitory phenomena of cough, sneezing, and general *malaise*, are usually more prolonged than in the other eruptive fevers. It does not always happen, however, that the functions of the mucous membrane are disordered, as well as the cutaneous surface. There are cases in which no catarrhal symptoms exist, and such cases are described as “*morbilli sine catarrho*.” Such cases occur during epidemics of the disease, and are but few in number.

Since the affection of the skin is uniformly present, while that of the mucous membranes is sometimes absent, the cutaneous eruption is necessarily the great characteristic of the disease; but the morbillous eruption being evanescent after death, we can only imperfectly trace its pathology. It first appears as a circular spot or blotch, similar to a flea-bite, slightly prominent, and scarcely sensible to the touch. Its color is of a pinkish red, or deep raspberry



THE FOLLOWING DIAGRAM REPRESENTS THE TYPICAL RANGE OF TEMPERATURE IN A CASE OF MEASLES. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE FOURTH DAY OF THE DISEASE:



During the period of incubation, slight feverishness, depression, and catarrh are present in adults; but when the disease is expressed, its commencement may be marked by violent shivering, or merely by chilliness, characteristic of catarrhal fever. The inflammation of the mucous membrane, of the eyes, and nasal fossæ, indicated by more or less constant sneezing, generally commences either with or before the primary fever, and consequently precedes the eruption by some days. This inflammation is perhaps, for a few hours, confined to fixed spots, and is marked by itching at the mucous orifices; then it becomes diffuse, and quickly changes to the serous; for a profuse watery discharge from the eyes and nostrils shortly follows, technically termed "coryza." This affection usually continues till the decline of the eruption, and in some cases to a later period. Children, as a rule, are not anxious to seek their beds; even with a temperature of 104° Fahr. they are still able to remain up; but in cases of pneumonia, with the same temperature, they desire to lie down at once.

The temperature rises rapidly towards the breaking out of the eruption. Its rise is steady to the *fastigium*, but if remissions are marked, they occur in the morning. If the fever is high before the eruption for many days, it indicates a severe case, and is apt to be attended with such nervous derangements as are indicated by somnolency, jactitation, or delirium. The *fastigium*, or maximum of temperature, generally coincides with the period of the eruption; and simultaneously with this increase of the fever the nervous



#### CHARACTERISTIC

rash. Blood in small quantities. In 1854 the recovery was the greatest (P).

**Symptoms.**—The symptoms of the consecutive local lesions are extremely few, for no without the secondary or tertiary is supposed sometimes to be cutis, and to exhaust itself *sine catarrho*." The varying physicians to consider the primary—namely, the "*morbilli mitiores*."

The primary fever may not last for a few days with symptoms of the latter is the case; but in no afterwards prolonged, and as of great intensity. Although the severity of the local lesions, yet an instance is known of the patient being overwhelmed or destroyed by the general depressing action of the poison, as is the case in typhus fever or scarlatina. The depressing powers of the poison, however, are considerable, and are often sufficient to confine the patient to his bed for a few days, and to leave him for a short time after the disease has subsided, weak and debilitated. The type of the fever of measles consequently greatly differs from that of typhus or of scarlatina, and the formidable brown tongue, so grave a symptom in the latter, is hardly known in the former, or only seen in a few fatal cases.

#### *Morbilli Mitiores.*

The essential characters of this affection are, that the poison produces primary fever, and a specific inflammation of the skin and mucous membranes,—the defervescence of the fever taking place while the eruption fades.

The symptoms may be divided into three stages: the first embraces the primary fever, or the period before the eruption, and may last from three to five days; while the second stage embraces the period of the eruption, and lasts from six to seven days. These two stages very commonly comprise the whole disease, whose usual course is then from nine to twelve days. The third stage includes any inflammatory action which may be caused by the tertiary action of the poison, and only occasionally exists.

The early symptoms of the primary fever are seldom severe, and greatly resemble those of an ordinary acute catarrh. They are, shivering, alternated with heat, frequent pulse, headache, derangement of the bowels, sometimes accompanied by nausea and vomiting; and these affections are so considerable that the patient usually takes to bed. At the end of a few hours the fever becomes continued, and the specific action of the poison commences by the mucous membrane of the eyes and nose inflaming, so that the light is painful; the senses of smell and taste are lost, followed by a copi-



The eruption is sometimes greatly delayed from causes not quite manifest. Excessive purging is thought to have this effect, or anything which greatly debilitates the system, hereditary or acquired unhealthiness of constitution, or the peculiarly malignant nature of the disease. The occurrence of the eruption is therefore to be looked for with anxious care, as the appearance of it, even though late, is in itself a favorable indication.

If the eruption suddenly disappears, or "goes in," it is no less an unfavorable omen, and is apt to be followed by dangerous results, diarrhoea, dyspnoea, coma, convulsions, all which unfavorable signs may again disappear on the reappearance of the eruption.

### [*Camp Measles.*]

Measles is one of the most formidable of camp diseases, and prevailed epidemically to a great extent in the United States and Confederate armies during the war of the rebellion. It happened chiefly amongst the regiments just organized at the State depots, and regiments in camps and barracks soon after muster into service, and young recruits. The epidemics occurred in the fall, winter, and spring. The disease was much more common in the regiments raised or recruited in rural districts than in those from the large towns, its subjects not having been previously exposed to its contagion. The mortality rate was large. In the general field hospital at Chattanooga, it was 22.4 in 100 cases. In General Hospital, No. 1, Nashville, it was 19.6 in 100, or nearly 1 in 5 (BARTHOLOW). Many died from the sequelæ, or became permanently disabled. Contagion from a specific poison, acting upon unprotected persons, was unquestionably the means of propagation of the disease. Dr. Bartholow states: "In one regiment which came under my observation, every man contracted measles who had not had it in early life. This was the rule in all regiments exposed to the poison. This statement is supported by the fact, that in 100 cases [analyzed], 91 had not suffered at any period of life from measles, and only 9 supposed that they had had the disease, but were not at all certain" (*Contributions relating to the Causation and Prevention of Disease, and to Camp Diseases, United States Sanitary Commission, 1867*).

The symptoms, intercurrent disorders, secondary affections, and morbid anatomy, of Camp Measles differ in no essential particular from those met with in the disease amongst civilians.

The great losses to the service from the disease, both by death and discharge from its disqualifying effects, make it a matter of moment to adopt, when practicable, the means of securing armies from these results. The value of the soldier is much increased by his having gone happily through an attack. The protective measures are: (1.) Exposing to the contagion at a favorable season, and under the best hygienic conditions possible, those who may be liable from previous exemption. (2.) Isolating the cases of the disease when they occur, and preventing exposure of those obnoxious to the specific poison.]

**Diagnosis.**—The diseases with which measles may be confounded are scarlet fever and some forms of syphilitic eruptions. The diagnostic symptoms between measles and scarlet fever are numerous; for there are many differences, both in the general course of the fever, the ranges of temperature, and particular symptoms of these





tends to be of the asthenic type, and is not unfrequently preceded by diphtheritic inflammation of the fauces, which gradually passes down to the larynx.

Diarrhœa is another danger to be encountered. During convalescence there is a tendency to looseness of the bowels, but which, if moderate, ought not to be counteracted, as it is commonly rather advantageous; but if suffered to continue, the consequences may be fatal.

Catarrhal ophthalmia, if the constitution be strumous, must also be watched for, and, if possible, prevented.

Measles, in any of the malignant forms described, is highly dangerous; and the danger is greater in the old than in the young—in cold than in warm weather.

**Causes.**—Measles were first noticed at the same time and in the same country with scarlet fever, and the two diseases have subsequently followed nearly the same course. They now prevail all over the world, are little influenced by season, are believed to be constantly in existence somewhere, and occasionally epidemic.

Measles, though incidental to every period of life, are most frequently contracted in childhood, when it is difficult to trace the effects of accidental circumstances, so that our knowledge of the predisposing causes is most imperfect. Both sexes, however, appear to be equally liable to this affection. With respect to the influence of season, it is generally supposed that measles break out most readily in the beginning of winter, increase till the vernal equinox, and then tend to subside towards the summer solstice. The deaths, however, from this disease, registered in England and Wales, show that the influence of season is exceedingly trifling.

**Propagation of the Disease by Direct Communication and Infection.**—It is admitted by all authors that a patient laboring under measles generates a poison which may be communicated directly, or which may contaminate the atmosphere with an impalpable poison. Like scarlatina, measles is thus eminently communicable; and in like manner no susceptible person can remain in the same room, or even in the same house, with an infected person, without hazard of taking the disease. In the year 1824 it was imported into Malta by some children belonging to the 95th regiment, and spread extensively in that island, so that many natives died. This circumstance was the more remarkable, as measles had not been in the island for many years. The *infecting distance* of this poison, it will be plain from what has been stated, must be considerable; indeed, it is often very difficult to isolate the disease in public schools, or other large establishments, where it sometimes appears.

The fact of measles being communicable has often been proved; but some difference of opinion exists as to the possibility of communicating the disease by inoculation. Healthy children have been inoculated either by blood drawn from the arm of a patient suffering from measles, or with serum taken from the vesicles which are occasionally found intermixed with the eruption,—an experiment which appears to have been first made by Dr. Home, with a view of producing a mild disease; but as no such result has been obtained,



Measles will not bear exposure of the surface of the body to cold so well as either scarlatina or small-pox, on account of the great tendency to bronchial and pulmonary inflammation. Children must therefore be watched night and day to prevent them lying uncovered, and special care must be taken to avoid exposure to cold during convalescence. In the *morbilli mitiores* the cough, the frequent vomiting, and the heavy catarrhal symptoms which so generally attend the primary fever, render medical attendance necessary from the first moment of the attack. The treatment of these symptoms, however, and also of the eruptive stage, as long as the patient continues free from any serious inflammatory affection of the lungs, need not necessarily be active, it being sufficient to alleviate the cough, allay the vomiting, and check the catarrh by some of the large class of saline laxatives, linseed tea, or mucilaginous mixtures, to which *antimonial wine* may be added if necessary, as a diaphoretic, and to subdue high vascular action. In making a selection from these, the physician must be principally guided by the state of the bowels and the condition of the stomach of the patient. If the bowels be constipated, the milder purging salts, as the *sulphate of magnesia*, are to be preferred. On the contrary, if the patient be purged, and the vomiting distressing, a neutral mixture or effervescing draught will be found most beneficial. There are many persons in whom the cough and catarrh are the most urgent symptoms; and in such cases, if the stomach be quiet, the *liquor ammoniæ acetatis*, in half ounce doses, combined with *camphor mixture*, from its more powerful action on the skin, is an excellent substitute. Another remedy, equally or perhaps still more useful, is *ipecacuanha*, of which from one to two grains may be given every four or six hours. Some practitioners prefer antimony to ipecacuanha, but antimony appears, at least in large doses, to act in some instances perniciously on the lungs.

The treatment which has been specified is, in most cases, all that is necessary throughout the whole course of the disease; and the greatly extended experience of Willan hardly enabled him to enlarge it. He was of opinion, however, that an emetic, given on the second or third evening, *somewhat* alleviated the violence of the catarrhal symptoms, and contributed to prevent the diarrhœa which usually succeeds measles. An emetic is especially useful if the disease be threatened with croup as a complication. During the eruption, he adds, "I have not observed any considerable effect from antimonials or other diaphoretics." Bathing the feet every evening seems a more beneficial application. Emulsions and mucilages afford but a feeble palliation of the cough and difficulty of breathing. With respect to opiates, they are not generally advisable: in the early stages especially, according to Willan, opium produces an increase of heat and restlessness, without conciliating sleep.

The catarrhal symptoms are frequently accompanied, even in the very earliest days of the disease, with much bronchial inflammation, and sometimes with pneumonia; or these affections may occur at any later period, after the decline of the eruption, from the tenth to the twelfth day of the attack. Although experience has shown



When convulsions occur in children, hot foot baths sometimes give relief, as well as sinapisms to the limbs; after which, if they do not subside, blood must be taken by leeches from the temples; and it is in all cases necessary to determine the most probable source of the irritation, giving rise to the convulsions—*i. e.*, whether they depend upon the specific poison of the disease, upon dentition, or upon intestinal irritation or cerebral disorder. Diarrhœa should not be checked suddenly, but kept under control.

### SCARLET FEVER.

LATIN, *Febris rubra*; FRENCH, *Scarlatine*; GERMAN, *Scharlachfieber*; ITALIAN, *Febbre Scarlatina*.

**Definition.**—*A febrile disease, the product of a specific poison, which is reproduced during the progress of the affection. On the second day of the illness, or sometimes later, a scarlet efflorescence generally appears on the fauces and pharynx, and on the face and neck, which spreads over the whole body, and commonly terminates in desquamation from the fifth to the seventh day. The fever is accompanied with an affection of the kidneys, often with severe disease of the throat, or of some internal organ, and is sometimes followed by dropsy. The disease runs a definite course, and as a rule occurs only once during life.*

**Pathology and Symptoms of the Disease in its Varied Forms.**—After a definite period of latency, the peculiar poison of scarlet fever induces a disorder of the blood, which is, in the first instance, made manifest by a *febrile state and a disturbed condition of the great nervous centres*. The primary fever having lasted for one, two, or three days, does not entirely subside, but the secondary actions of the poison are set up as a peculiar eruption, preceded, followed, or accompanied by a sore throat. The eruption runs a course of from six to eight days, but the duration of the affection of the throat is more indefinite, and varies from eight to twenty, or more days. The fever continues during the eruption, and as long as the sore throat exists; but this being terminated, it subsides, and the disease is ended. In a few instances, however, tertiary results succeed, as dropsy or inflammation of the joints, diseases quite as formidable as any which had preceded them. As in ordinary fever, the poison of scarlet fever acts on the brain and its membranes, often causing the usual forms of inflammation of those parts, modified in their course and effects by the nature of the specific febrile disease.

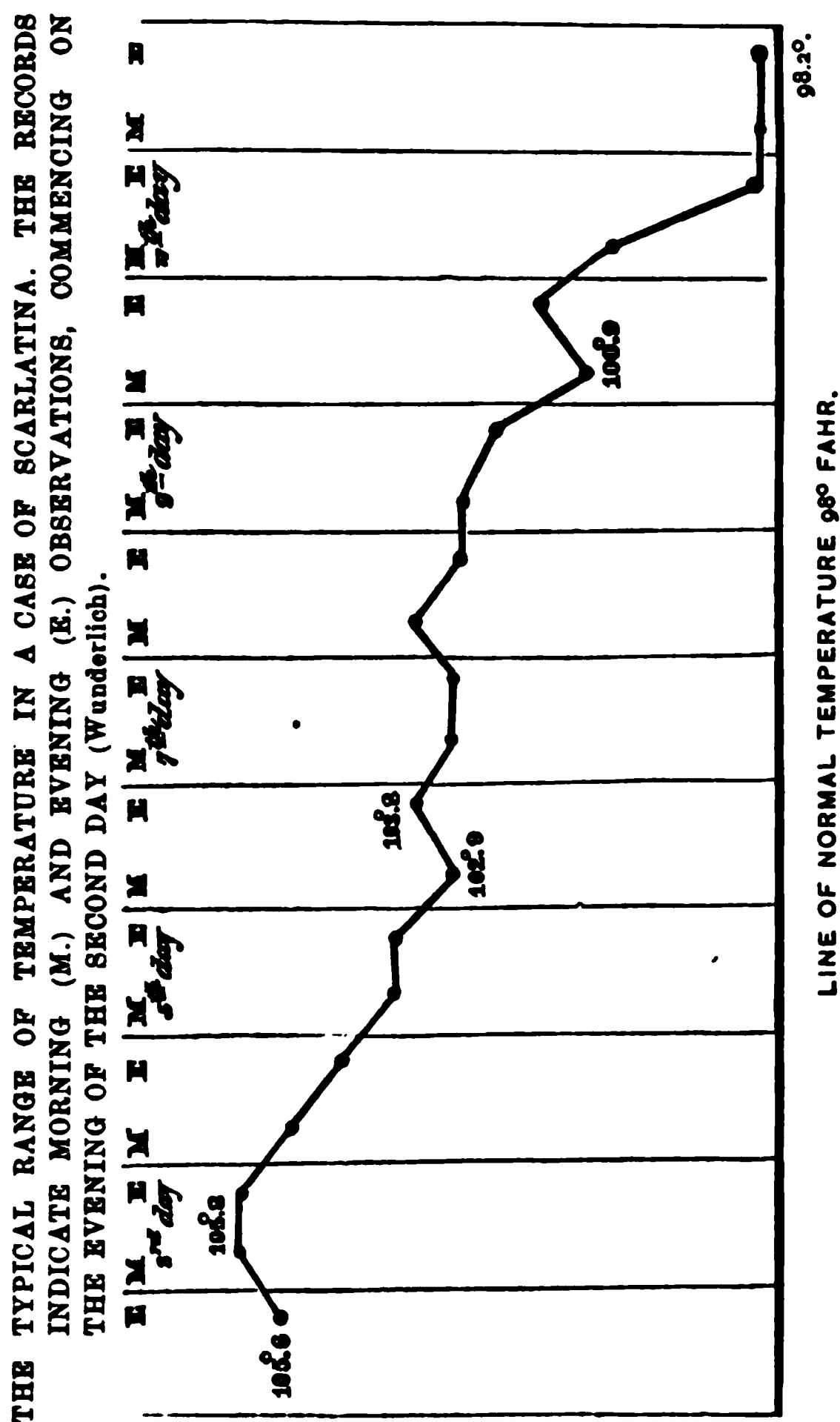
That fever precedes the specific actions of the skin in this disease is so general a rule that it has few exceptions; and the pyrexia has been occasionally so severe as to destroy the patient before the more specific lesions of the disease have been set up. Dr. Andrew Anderson writes that he has seen death take place in six hours from the commencement of the disease—the child, in fact, dying poisoned (*On Fever*, p. 77). In suddenness of danger it thus ap-



affection follows a slowly progressive course; (3.) A period of resolution (HAMBURGER). Such cases sometimes terminate by uræmic symptoms and convulsions. An unusual case of this nature has been recently recorded by Biermer. It happened with a boy five years and a half old, and ended fatally on the thirty-fifth day. No urine was passed for 108 hours between the twenty-first and the twenty-sixth days of the disease, and extremely little for five days more. Yet during these ten days there were no uræmic symptoms, nor any notable dropsy. The uræmic symptoms first set in after the urine began to be secreted freely, and it was but slightly albuminous (*Syden. Society Year-Book*, 1861, p. 218). Occasionally the *squamæ* of the cuticle are so large as to preserve entire the whole epidermis of the palms of the hands and of the soles of the feet. Frequently, however, the material of desquamation is furfuraceous or scaly. Frank has seen the cuticular *squamæ* come away with the hair, nails, and even with *verrucaæ* attached. In a few instances the termination is by ulceration and sloughing of large portions of the integument.

Whatever may be the color or description of the eruption, it does not attack all parts of the body simultaneously, but appears partially, or in a succession of crops, the order of which may be stated as follows: On the first day it spreads universally over the face, neck, and upper extremities; on the following day over the trunk, but is less general on the back than on the abdomen; and, lastly, on the third day, it has extended itself over the lower extremities. The duration of each crop is about three days, when it disappears, and in the order of attack, falling from the head and upper extremities on the fourth day; from the trunk on the fifth day; and from the lower extremities from the sixth to the eighth day. The order of attack, however, which has been mentioned is not constant, for in some few instances the eruption appears first on the trunk and lower extremities, and only on the second day very faintly on the face and upper extremities. The disease attains its height, and the fever maintains its course, usually from the fifth to the ninth day, when, in favorable cases, continuous defervescence sets in, and all the symptoms begin to decline. The fever does not subside on the appearance of the rash, as is the case with small-pox, but continues with various degrees of violence and ranges of temperature, throughout its progress. The pulse is often 120 to 130 in a minute, and sometimes beats with considerable force. The skin frequently indicates by the thermometer a temperature of 105°, 106°, or even 112° Fahr.; and it is dry, with a sensation of burning heat till about the third day, when the maximum of temperature is attained. From the third to the ninth day the range is maintained between 103.8° and 102.9°, and begins to subside about the tenth or twelfth day, after which the defervescence is continuous. The difference in these respects between scarlatina and measles may be appreciated at once by glance at the account and the diagram given of measles, and comparing it with the following, which shows





There is no remarkable increase of fever heat preceding complete defervescence; and after the *exanthema* has reached its maximum, the decrease of temperature proceeds by no means rapidly. The commencement of the decrease may be marked by a few decided and rapid falls; but its farther fall is decidedly lingering, and is even sometimes interrupted by small increases of temperature, so that the whole process of defervescence occupies, as a rule, from five to eight days. It is only in very mild or anomalous cases that the temperature rarely exceeds 101.8° Fahr., and these cases sometimes show a rapid defervescence, completed in a single night. It is essentially a short fever, the range of temperature, according to Dr. Sidney Ringer, forming cycles composed of a variable number of days, generally of five; a fall of temperature taking place on the fifth, tenth, or fifteenth day of the disease (*Med.-Chir. Society Trans.*, 28th January, 1862).

The poison of scarlatina as frequently falls on the mucous mem-

brane of the eyes and nasal fossæ, and excites a similar eruption over those parts, as on the skin, at first consisting of a distinct punctated or dotted appearance, which changes in a few hours to one of diffused redness. The inflammation of the ocular membrane, however, has this peculiarity, that it does not distress the sight, for the eye bears light without inconvenience, and in no case is it combined with coryza. Neither is sneezing a consequence of the affection of the nasal membrane; and only in a few severe cases is there any discharge from the nostril. As the eruption attacking these parts generally appears with the first crop of the *exanthema* of the skin, so does it generally die away with the cutaneous eruption. This inflammation usually terminates by resolution; but in a few instances the alæ of the nose ulcerate, and sometimes mortify.

The lingual and buccal mucous membranes are also often the seat of a similar exanthema, presenting nearly the same appearance as in other parts. The papillæ of the tongue, however, are singularly elongated and enlarged, and stand up salient and erect, and of a deep scarlet color, above the thick, white creamy mucous fur which coats the lingual membrane; and hence the term "strawberry tongue," from the supposed resemblance to the exterior of a strawberry. The tip of the tongue is of a vivid red, through development of the papillæ. By and by the fur falls off, and the whole dorsum of the tongue is then left clean, red, and raw-looking. This affection lasts longer than that of the eyes and nose, and usually terminates by resolution, although, in a few instances, the buccal membrane ulcerates and mortifies.

The sore throat, or inflammation of the faucial membrane, though not so constant an affection as that of the skin, yet when it does exist, it is often of much longer duration, and is a much more grave disease. It may either precede all the other symptoms, or it may occur at any period of the fever. This inflammation, at first punctated, then diffused, usually runs into ulceration, and the character of the ulcer is so completely in unison with the state of the constitution as to enable us, according as it is slight or severe, to divide scarlatina into two great varieties—namely, into "*scarlatina mitior*" and "*scarlatina gravior*." The first, or sthenic form, is marked by a greatly enlarged or swollen state of the tonsils, which are of a vivid or bright red-color; and, when ulceration takes place, the ulcers are seldom deep, or the sloughs slow to come away, but usually they separate about the fifth or sixth day; so that in mild cases the sore throat is healed about the eighth or tenth day, and in more severe ones about the fifteenth or twentieth. In malignant cases, or in *scarlatina gravior*, the tonsils are much less tumefied and enlarged, but much more loaded with blood, and of a deeper, and sometimes of a livid color. The ulcers, also, are deep and formidable, and the sloughs are thrown off later in the disease. They are likewise slow to heal, or not to the end of three weeks, and in severe cases not till four or even six weeks have elapsed, during which period the fever continues, and the patient remains in considerable danger.

The inflammation of the throat is not limited to the tonsils, but



presence of albumen, without diminished secretion, is almost a regular phenomenon in the course of the disease, independent of dropsy, as shown by Dr. James W. Begbie, yet if the urine becomes highly albuminous and diminished in quantity, the dropsical complications may be apprehended (*Edin. Med. Journal*, Jan., 1849, and Oct., 1852).

More or less congestion of the kidneys occurs in every case of scarlet fever, although, like the sore throat, it may often be so slight as not to give rise to any prominent symptom (BEGBIE, ANDERSON). The scarlatinal dropsy is very generally considered as most intimately connected with the kidney disease; and when the kidney disease is well marked, the characters of the urine exactly resemble those in acute Bright's disease (PARKES). On the other hand, there is also evidence decidedly in favor of the opinion that albuminuria may be wanting in scarlatinal dropsy (see Parkes *On the Urine*, p. 264).

*The condition of the urine* in scarlet fever ought to be ascertained daily in every case, especially during the period of convalescence. "It is of more importance," writes Dr. Andrew Anderson, "that you should examine the urine than that you should feel the pulse of a convalescent from scarlet fever." The urine has the ordinary febrile characters. During the first six days the amount is small; the urea and uric acid are increased in amount, and sediments of urates occur. The chlorine is sometimes greatly lessened, and augments during convalescence. On the sixth to the eighth day, if the case goes on well, the urine becomes abundant, pale, and the reaction neutral or feebly acid. There is bile-pigment present during the first six days; and in a large proportion of cases, though not in all, the urine becomes albuminous. Dr. Warburton Begbie believes it to be present at some period in almost every case. It is usually associated with a large amount of renal, pelvic, and bladder epithelium, but not with renal cylinders (BEGBIE), unless there be dropsy. The albuminuria occurring during desquamation is usually transient; but it may continue till an attack of dropsy occurs—disappearing and reappearing, when dropsy comes on a fortnight or three weeks later. In malignant scarlatina, as in malignant variola, there may be considerable hæmaturia or passage of dissolved hæmatin (Parkes *On the Urine*, p. 263).

Intercurrent inflammations of the synovial membranes have been described by Withering, Sennertus, Heberden, and others. This disease may attack the wrist, ankle, or knee-joints, and usually terminates by effusion of serum; and in some cases the cavities of the joints contain pus. This inflammation seldom occurs till after the eruption has subsided, and is generally a tertiary phenomenon in the course of the specific disease.

Such are the morbid phenomena which have been observed in the ordinary course of scarlatina, and with sufficient constancy to mark the disease as due to a specific poison; but these appearances are only to be found when the disease is of moderate intensity and the patient survives some days. In severe and rapid cases the patient may die, not from any organic lesion, but from the intensity of the shock, in the first instance, on the nervous system; for Bre-



through the whole course of the disease. The fever, however, varies greatly in intensity, as already indicated, from a mere febricula to the severest forms of a typhoid type in protracted cases.

### 1. *Simple Scarlet Fever.*

This form is known by the name of *S. mitis* and *S. sine angina*. It is the simplest form of scarlet fever, and is limited to cases with the fever and eruption, without any affection of the throat, or to cases with "a scarlet rash, with redness of the throat, but without ulceration."

The symptoms of this variety are extremely mild, so that the patient is frequently not confined to bed. The primary fever, except that the pulse is rapid, is little more than a mere febricula, and is not aggravated on the appearance of the eruption. The eruption appears at the end of twenty-four or forty-eight hours, and the crops follow each other according to the usual order of succession, appearing first on the face and neck and upper extremities; on the following day on the trunk; and on the third day on the lower extremities, when the disease has reached its acmé. On the fourth day the rash begins to decline, and fades from the face, neck, and upper extremities; on the fifth day it disappears from the trunk; and on the sixth or seventh day it is evanescent over the whole body. The color of the rash is always more florid during the night than in the day; and on its declining, desquamation takes place. With the disappearance of the rash the fever of this variety ceases, and the disease terminates; but it often leaves the patient in a state of considerable debility for several days, and may be followed by albuminuria.

### 2. *Anginose Scarlet Fever.*

In this form of the disease the specific action of the poison is mainly limited to one region—that of the throat—the eruption on the skin being altogether wanting, or appearing at a later period than usual, generally by one day; and, as a general rule, is less copious and less diffuse than in the other forms. It is "a more severe form of the disease, with redness and ulceration of the throat, and a tendency to the formation of abscesses in the neck."

There is seldom a season in which scarlatina has been in any degree epidemic, that cases have not occurred in which patients (not having previously had scarlet fever) are seized with severe fever and sore throat, unaccompanied by any eruption, and who, on subsequent exposure to the contagion of scarlatina, have been found insusceptible of the action of the poison. Hence it is inferred that the disease they have passed through must have been a variety of scarlet fever, or *scarlatina sine eruptione*, making itself manifest by a peculiar sore throat, associated with the febrile phenomena.

This disease, therefore, essentially consists in fever and sore throat. It has been stated that the state of the throat is constantly in unison with the state of the constitution, and consequently this form of disease, according to its severity, assumes all the symptoms which



tertiary affections more frequent, and, consequently, the disease is more grave and the danger more formidable.

The more remarkable symptom which distinguishes this form of the disease is the state of the tonsils. In the milder form previously noticed, it has been stated that the tonsils are either slightly affected or greatly enlarged, of a bright red, and the ulcers comparatively superficial; but in this severer form the tonsil, though less swollen, is more gorged with blood, more livid in color, while the ulcers are foul, deep, and burrowing; the secretions of the mouth are more copious, and generally impregnated with the offensive sordes of the sloughs; while deglutition, if less difficult, is perhaps infinitely more painful, and the mouth often so tender that the slightest touch excoriates it. The ulcers likewise are slow to granulate, and only heal after a tedious treatment; and in the worst cases they spread in every direction, the parts tending to vesicate and even to mortify previous to the death of the patient.

*The eruption* offers some peculiarities, being often later by some hours in coming out, its color darker and more livid, its duration more uncertain, and its distribution more irregular and capricious than in the milder form. The primary fever, likewise, is usually longer, the delirium earlier, and the depression more complete than in the milder forms; and towards the close of the disease the tongue becomes brown, and the symptoms closely resemble those of the last stage of typhus fever.

Such are the more marked characters of the severer form of scarlatina; but it often happens that the progress of this disease (unless the range of temperature is regularly and continuously recorded) is silent, slow, insidious, scarcely marked by any prominent symptom till the degree in which the constitution is subdued by this formidable poison is shown by the inflamed nasal membrane discharging its fetid ichor, causing mortification of the alæ of the nose, or mortification of the lip or cheek; or it seizes on some remote part, as the toe, the leg, or the whole of a lower extremity, and which, for the most part, terminates the life of the patient. It may pass into the next form of the malady, namely,—

### 3. *Malignant Scarlet Fever.*

This form is that which is known as the “malignant sore throat,” or “putrid sore throat” of some authors; and is the name now generally applied to certain cases of extreme severity, into which some of the forms already described may pass, as if by insensible gradations. In this variety “the throat tends to slough; the scarlet rash is scarcely, if at all, visible, petechiæ are often seen on the surface, and the fever is of a low form.” In others, the violence of the attack is so sudden that the patient is at once struck down by the force or virulence of the poison, the type of the attack being at once septic, adynamic, typhoid, and malignant. The extreme severity of the constitutional symptoms is marked by the smallness, feebleness, and irregularity of the pulse; the oppressed, short, and quick respiration; the appearance of early raving, stupor, and some-





completely destroyed, the tympanic cavity becomes inflamed, ulceration of the membrane takes place, and perforation follows.

This morbid state is most difficult to remedy: a chronic discharge from the ear is established, which is of the most offensive kind, and which may continue till the whole of the internal ear is involved in the destructive and inflammatory processes; till the delicate and soft tissues in the cochlea and semicircular canals are destroyed, and the petrous portion of the bone itself dies; till the mastoid process, with its capacious osseous areolæ, becomes the seat of an obstinate carious process; or even till the brain itself, or the membranes, are involved in the unhealthy inflammatory process. Such a combination of effects occasions great and protracted sufferings, and sometimes in the end a fatal result (BRUCE, ANDERSON). A similar inflammation may destroy the tissues in the back part of the pharynx, extending towards the base of the cranium and upper cervical vertebræ.

A frequent form in which the tertiary actions of the poison of scarlatina are manifested consists in inflammation of the joints, and dropsy; and it is singular that these diseases are more often set up after mild than after the more severe forms of this fever. In such cases, about the time of the disappearance of the rash, the joints of the wrists or fingers, of the knees or other articulations, become swollen and inflamed, and present all the phenomena of an attack of acute rheumatism. This affection keeps up the fever, and prolongs the whole duration of the disease for many days beyond the usual period.

Again, in a given number of cases, not exceeding three per cent. in general, but in different seasons, or under different treatment, sometimes amounting to twenty per cent., the tertiary action of the poison produces dropsy. This affection usually occurs about the twenty-second or twenty-third day, or about the time when the patient is convalescent, and more often after a mild than after a severe disease. Dropsy more commonly begins with pallor of the countenance, and with œdema of the face; then the hands and feet swell, and, in a few cases, the areolar tissue of the trunk and lower extremities becomes enormously distended. When the areolar tissue is thus slightly or more generally distended with fluid, effusion may take place into the cavities of the head, chest, or abdomen. When the brain is threatened, the effusion is commonly preceded by the usual hydrocephalic headache, by convulsions, and sometimes by blindness. Effusion into the cavity of the chest or of the abdomen causes the usual symptoms of hydrothorax and of ascites, which have been described. In the former instance, however, the watery fluid is sometimes poured out so rapidly as to destroy the patient in a few minutes or in a few hours.

The first appearance of the œdema or effusion is usually preceded or accompanied by an accelerated pulse, by the urine being scanty, commonly turbid, and passed with pain: the quantity, however, is shortly increased; and if examined when passed copiously, it is found to be of low specific gravity, or from 1.011 to 1.017, and to contain albumen, sometimes blood, renal epithelium, and cylinders.



tions to the statement that an attack of scarlatina gives an immunity from a second attack. Dr. Binns has seen instances of scarlet fever occurring twice in the same person, while Sir Gilbert Blane met with an instance of its occurring thrice in a young lady, without the least suspicion of ambiguity or possibility of mistake in diagnosis. Dr. B. W. Richardson shows that it may recur once or even twice in the same person. But these events are rare; and death from a second attack is unknown as a fact.

Scarlet fever has often coexisted with the vaccine disease, and with erysipelas, and this poison is consequently capable of coexisting in the system, not only with those that have been mentioned, but probably with all other morbid poisons.

The poison of scarlatina is absorbed by the mucous membranes; and absorption is also evident from the fact of inoculation having been effected through the skin. Children have been born laboring under this disease.

The period of latency varies from a few hours to ten days. In one case inoculated by Rostan the disease appeared on the seventh day; and the specific poison is probably capable of communication from the patient to others as soon as the primary fever has formed, and perhaps continues to be so till the sore throat has perfectly healed, supposing that affection to continue after the eruption has died away.

**Prognosis.**—The mortality from scarlet fever varies greatly according to the season, and also, perhaps, according to the fatality of the epidemic. In some years the proportion of deaths is not greater than three per cent.; but Sir Gilbert Blane says his practice gave one in four. He was consulted probably only in the worst cases, for in the same year it appears, from the reports of other practitioners, the deaths varied from one in six to about one in thirty.

There is perhaps no disease in which the progress is so capricious: for it is found to vary with the several forms, types, complications, epidemic constitution, and with the treatment in a most remarkable degree. The mortality is greatest in the period of infancy and childhood—from one to five years. In relation to mortality, it seems second in this country as to severity, typhus fever standing first (RICHARDSON). It is twice as fatal in towns as in the country. "There is one condition in which the disease is almost invariably fatal; that is the puerperal state. No precaution ought, then, to be neglected, no precaution ought to be thought excessive, which tends to prevent a woman from receiving the poison of scarlatina while pregnant or recently delivered" (DR. ANDREW ANDERSON). Fever during the pregnancy most certainly ends in abortion and death. If the woman be recently delivered, the disease will be of the most malignant type, and almost always fatal.

**Treatment.**—Scarlet fever being evidently accompanied by many highly inflammatory symptoms, the practice of bleeding was adopted on the first breaking out of the disease, in all countries, and, according to Willan, *with the most disastrous results*. This mode of treatment was adopted by Morton; and he speaks of witnessing 300 deaths from scarlatina in a week. It prevailed down to the time of Huxham, who abandoned it, and introduced a treatment by bark. In



As soon as this object is effected, and it is ascertained that the tonsils are still greatly enlarged and swollen, the practice (supposing the patient to be *an adult*) is to relieve them by the application of six to twelve leeches to the throat; and the bleeding may be further encouraged by the application of a poultice. The trifling loss of blood thus sustained does not impair the general strength of the patient, if it is done sufficiently early, while it greatly reduces the swelling of the tonsils, and may prevent them becoming permanently enlarged. Another advantage is gained by the application of leeches to the throat—namely, that they relieve the affection of the head; for we constantly observe that, in diseases depending on morbid poisons, the head symptoms are relieved by relieving the part specifically acted upon.

The tonsils having been thus relieved, the fever ought to be permitted to run its course uninfluenced by medicine, the patient being only refreshed by the occasional administration of the saline draught, so grateful to his parched mouth and feverish state. If stimulation be adopted in these cases, we are apt to bring back the tumefaction of the tonsils; while, on the contrary, if we take more blood, we hazard producing the more serious accidents incident to scarlatina. The medicines, therefore, that have been mentioned should be persevered in till the disappearance of the eruption, and till the healthy granulations of the throat, and the decline of the fever, give certain evidence of a state of convalescence. At this point some mild tonic medicine is desirable, and prepares the patient once more for the fullest enjoyment of health. This is the most successful mode of treating cases of scarlatina in its milder forms. With children, however, it is better to trust to the soothing effects of warm poultices round the throat, than weaken the child by loss of blood.

The severe forms are characterized by the less swollen state of the tonsils, and by their being more livid and gorged with blood; by the ulcers being deeper and more spreading; and by the slough being fouler than in the milder varieties. As there is a greater tendency of parts to run into mortification, the necessity of adopting a more stimulating plan of treatment, and one more calculated to support the powers of the constitution, is manifest, and experience has shown this view of the case to be correct. The administration of wine, and of the "*extractum carnis Liebigii*," should therefore be the basis of the treatment of such cases. The quantity of wine for an adult may be from four to six ounces in twenty-four hours, and for a child about half that quantity. The wine may be either port or sherry, and should be drunk in small quantities, mixed with two-thirds water; or it may be given with sago, arrow-root, jellies, or other slops. The earlier the wine is given in the disease the better, and when delirium does or does not exist; regardless, also, as to whether the tongue is moist and white, or brown and dry; and it should be continued till the patient is decidedly convalescent. Liebig's extract of flesh should be given like beef-tea, as a drink. While pursuing this plan, it is necessary that the patient's bowels should be attended to. The treatment by wine is often ex-



fed; and preparations of iron may be given with advantage. The *muriated tincture* seems to have the best reputation; and the *iodide of potassium* in small doses is also useful. With this latter remedy the *syrup of the iodide of iron* may be combined, if it is desirable to continue the *chalybeate*, or syrup of the phosphate of iron in drachm doses.

Blisters, as a means of relieving the throat, are unnecessary, and are better omitted.

Gargles are unnecessary for children, for they cannot gargle; but they are of the greatest service, especially the deodorizing gargles or washes, when the patient can be taught to use them. A weak solution of chloride of lime, or of chlorine water, or of Condyl's fluid, or of the permanganate of potash, is well adapted to such a purpose. But the following is recommended as the most effectual gargle:

Solution of peroxide of hydrogen (containing ten volumes of oxygen), six ounces; tincture of myrrh, an ounce; rose water, five ounces (RICHARDSON).

This gargle may be used at pleasure: it is refreshing to the patient, and removes the offensive secretions readily. In the case of young children, who are unable to use a gargle, the throat may be washed out, by holding the little patient with the face downwards, and by pumping the solution over the surface of the fauces through a bit of gum catheter from a double-acting India rubber bag (Richardson, *Clinical Essays*, p. 110). As an invariable routine practice, Dr. W. T. Gairdner strongly recommends that "*the patient inhale the steam of hot water from the beginning to the end of the fever; as long, at least, as the throat is sore.*" In slight affections it is sufficient to employ infusion of linseed in water, acidulated with nitromuriatic acid, weak solutions of alum, nitre, or common salt. When membranous diphtheritic patches are observed on the fauces, and the color of the mucous membrane is of a dark red, capsicum infusion, or powdered red pepper, is an excellent application (WOOD); and in children who cannot gargle, it may be applied with a hair pencil. Solution of zinc, and nitrate of silver, are also of service.

These details are given because the physician must decide, upon the merits of the individual case, the nature of the treatment he will adopt. But it must be remembered that cases of scarlet fever, if left to themselves, with rest and careful nursing, will generally get well. The mere intensity of the fever is no ground for active interference by way of treatment, if the pulse is full and of good strength. Much is to be trusted to the shortness of the fever, remembering that there is no disease in which the patient is more apt to be delirious, with less danger, than in scarlatina (W. T. Gairdner, *Clinical Medicine*, l. c.).

**Dietetic and Preventive Treatment.**—The diet of the patient should be slops, light nutritious broths, and jellies. Fumigation will not, it should be remembered, destroy the miasmata in the sick-room; and, consequently, the doctrines of cleanliness, of ventilation, and of separation, are as imperative in this disease as in small-pox. We





The Arabians first described them, and considered them as varieties of one and the same disorder. Many essential differences, however, were soon observed to distinguish small-pox; but the points of resemblance between measles and scarlet fever were so many that it was not until fatal accidents had occurred, from great error in confounding them, that their differential characters were remarked, and their separate identity established. Now it is a generally received doctrine that measles and scarlatina, in their essence and in their symptoms, present two well-defined states of disease. This is, indeed, one of the most indisputable facts in Pathology. By Schönlein, measles has been classified as a peculiar exanthematic form of catarrh; and scarlatina is placed amongst the group of erysipelatous diseases; while, according to the experience of Dr. Küttner, of Dresden, there are "androgynous" cases calculated to embarrass the most experienced "diagnostiker." Measles and scarlet fever were especially confounded under the common name of *morbilli*; and even as late as the middle of the eighteenth century, writers of the highest repute supported the identity of measles and scarlet fever (the *morbilli confluentes* of Sir William Watson).

All authors before Sauvages (1768) had used the term *morbilli* (the term now in use) to designate measles; but he adopted a new name, and called measles by the designation of "rubeola"—an innovation which has caused much confusion, having been adopted by some (such as by Willan and Bateman) and rejected by others. Hildebrand, following the old nomenclature, calls measles *morbilli* and scarlet fever *scarlatina*; and terms the disease now about to be considered *rubeola*, as has been done by Dr. Copland. The German authors call it *rötheln*, and by this name it was first described by a most distinguished and learned Scotch physician, Dr. Robert Paterson, of Leith, in 1840. He is the only physician in this country who has given an original description of the disease in the English language, his description of the disease being drawn from many cases of it which occurred in his practice.

A difference of opinion prevailed amongst authors as to whether or not this disease is of a distinct and specific form. Those who have most recently described it (Hildebrand, Paterson, and Copland) consider it to be a disease possessing characters common to both measles and scarlet fever, as well as characters peculiarly its own. In truth, it seems to be a hybrid disease, developed from combined poisons of the two fevers, measles and scarlet fever. Dr. Küttner, of Dresden, states that he has seen occasionally in the same individual portions of the skin presenting the scarlatina eruption, while in other parts the eruption of measles was to be seen. He thus recognized not only examples of transition, but he recognized cases which may be termed hybridous (*Dublin Hosp. Gazette*, 15th Dec., 1858; and Ranking's *Abstract*, vol. xxix, p. 20).

**Symptoms.**—The febrile stage of the disease varies, like all the diseases already noticed, not only in the severity of the symptoms, but also in the length of the attack when compared with scarlet fever. It usually commences with rigors, not severe, but continuous. More or less cough soon makes its appearance—of the same clang-



doubly severe by the state of the throat. The pulse is very frequent; the skin hot and dry; and there is great restlessness, expressed by children tossing the head frequently from side to side, accompanied with frequent starting; and they are sometimes seized with convulsions. It is in this stage, in the worst forms of the disease, that death generally occurs, and that by coma. It may, however, take place either by suffocation from the large quantity of vitiated mucus, or by convulsions and subsequent coma. Vomiting is an occasional symptom during this stage, and, like convulsions, is sometimes seen in mild cases of the disease in children.

The eruption in mild cases, in general, continues distinct for from four to five days, during which time the other symptoms are going on favorably, becoming gradually milder as the period of the decline of the eruption draws near. In severe cases, however, the rash keeps its bright color and distinct form for a much longer period—*e. g.*, six, eight, or ten days.

The termination of the eruptive stage is, in some instances, marked by what is termed a distinct crisis,—such as the occurrence of copious sweating, deposits from the urine, diarrhoea, and epistaxis. Most commonly, however, there is no such crisis, but the eruption gradually fades, and the disease subsides.

As this happens, the desquamation by furfuraceous scales gradually ensues. This event is indicated by the appearance of scales towards the centre of the patches of eruption, to the margins of which they gradually extend, and soon spread over the whole body. The scales are small, and not unlike those of measles. On the hands and feet the scales are larger, but never reach the size of those of scarlet fever (ROBERT PATERSON).

**Lesions Seen in Fatal Cases.**—The accounts of these are few in number. They vary according to the period of the disease at which death occurs. Death most frequently happens during the eruptive stage, from coma, or from the affection of the throat and lungs. No morbid appearances of a uniform nature can be observed connected with the mode of death by coma; but when death happens from pulmonary oppression, the lungs are found much congested, the mucous membrane of the bronchia injected, with a copious mucous secretion. The throat presents very similar appearances to those which are seen in scarlatina,—great tumefaction, and dark coloration of the membrane lining the throat, dark aphthous spots, and large quantities of vitiated viscid mucus.

**Diagnosis.**—The accompanying febrile symptoms at once distinguish the disease from roseola, as also do the peculiar characters of the eruption. The only other affections with which it may be confounded are measles and scarlet fever. The following table points out the diagnostic marks more clearly by contrast than can otherwise be done, and shows that rubeola, rötheln, or the mixed disease, has every right to be considered as a distinct affection:



*Symptoms accompanying the Eruption.*

The symptoms which accompany the eruption in each of the three diseases are quite the same as those of the premonitory fever. It is proper here, however, to remark, that it is only in the anginose and malignant varieties of scarlatina that we have sore throat, there being little or none in the simple scarlatina, while in the mildest kind of rōtheln this is always a prominent and troublesome symptom.

## SCARLET FEVER.

## RUBEOLA OR RÖTHELN.

## MEASLES.

*Desquamation.*

The cuticle in this disease is thrown off in patches of considerable size, the largest being from the hands and feet.

The desquamation of rōtheln consists of minute portions of cuticle, like scales of fine bran.

The desquamation always begins towards the centre of the eruptive patch, and gradually extends to the circumference.

The desquamation of measles consists of minute portions of cuticle, like scales of fine bran.

*Sequelæ.*

Anasarca is the most common sequela of scarlet fever. It is extremely common, and most frequently occurs after the mildest cases; swelling and suppuration of the cervical glands is also common.

"I have noticed one case of dropsy after a mild, though well-marked attack of this disease; swelling and suppuration of the cervical glands also frequently takes place." (DR. PATERSON).

Affections of the lungs and pleura; tedious distressing cough; chronic bronchitis; pneumonia tubercles; gangrenous inflammation of cheeks, gums, lips, genital organs, &c.; dropsy occasionally occurs, but very rarely; diarrhœa is very common after some epidemics.

**Prognosis.**—It requires to be as guarded as in scarlatina; for, like scarlatina, rubeola is often an extremely and rapidly fatal disorder. The greater or less acuteness of the premonitory fever generally affords us a means of judging as to the probable severity of the eruptive disease; and in general it is a mild disease. To have a copious secretion of mucus in the back of the throat is always a bad symptom, or regurgitation of fluids by the nose. The chest ought to be examined from day to day, as sudden inflammatory action is apt to be established, and often it rapidly proves fatal. The condition of the urine requires also to be daily investigated.

**Treatment.**—The treatment is similar to that of scarlet fever. The functions of the skin are if possible to be stimulated; and Dr. Paterson found that the *aqua acetatis ammoniæ*, in the proportion of two ounces to half an ounce of antimonial wine and four ounces of water, made into a mixture, was the most useful agent. The use of colchicum was also had recourse to with decided benefit.

## DENGUE.

LATIN, *Denguis*; FRENCH, *Dengue*.

**Definition.**—A peculiar febrile disease, commencing very suddenly, and conjoined with severe pains in the joints, which swell; succeeded by general heat of skin, intense pain in the head and eyeballs, and the



or seventh day the eruption appears in the form of a scarlet efflorescence on the palms of the hands, which spreads rapidly over the body, and gives relief to the symptoms of febrile irritation. The eruption is extremely variable in character, being sometimes smooth, red, and continuous, as in scarlet fever; sometimes in patches, rough, and of a dark hue, as in measles; and occasionally either papular, vesicular, pustular, or furunculous; often with a mixture of two or more of these forms. The complaint gradually subsides, and leaves the patient with some rheumatic stiffness or soreness for a longer or shorter period, with feelings of weakness and mental depression. The duration of the affection varies with the length of the remission; but on the average is about eight days. Decided implication of the mucous membrane of the mouth and throat prevailed in the last epidemic in Calcutta, with an almost entire absence of the articular pains.

**Treatment.**—Emetics and purgation subdue the fever; but as the disease runs a specific course, time is an essential element in the treatment. Calumba, rhubarb, and soda form a useful alterative medicine. Ophthalmia is sometimes consequent on this disease, and is to be subdued by leeches to the inner membrane of the eyelids. Palliation and alleviation of symptoms, as they arise, chiefly by opium and alkaline remedies; following the indications given under “Scarlatina” and “Rheumatism.”

### ERYSIPELAS.

**LATIN,** *Erysipelas*; **FRENCH,** *Erysipèle*; **GERMAN;** *Erysipelas*—Syn., *Rothlauf*; **ITALIAN,** *Risipola*.

**Definition.**—*A febrile disease, associated with a peculiar eruption of the skin. The inflammation which attends this eruption is apt to spread indefinitely, and may involve the areolar tissue beneath the skin.*

**Pathology.**—As in other diseases of the miasmatic order, it is believed that in erysipelas a specific poison is absorbed and infects the blood, and that after a given period of latency it produces fever. The specific action of the poison, however, is mainly made manifest by inflammation of the skin and subcutaneous areolar tissue, which runs a definite course, and sometimes terminates in inflammation of the membranes of the brain. The inflammation and the fever are of a peculiar nature, not yet clearly understood. In Scotland the disease is known by the name of the *Rose*; in England it is sometimes called *St. Anthony's fire*.

Idiopathic erysipelas is very constantly preceded by fever—eighteen times out of twenty—and although it may be supposed that the fever is consecutive to the inflammation of the skin, yet before the redness of the skin is seen, the temperature, if measured by a thermometer, will be found above 98.6° or 99° Fahr., and attended with general malaise. The affection of the areolar tissue may be trifling, but it is seldom altogether wanting.

The pathological phenomena which result from the action of the





suppurative process extends between the muscles, causing extensive and often irreparable mischief. In the event of this inflammation terminating by gangrene, the integuments of an entire limb are sometimes detached, laying bare the muscles, a large artery, or a bone, involving the aponeuroses and tendons, and sometimes destroying the interior of a joint. Gangrene, however, does not equally take place in all parts, for it is seldom seen on the scalp, the face, or the trunk. It is the extremities, then, and more especially the leg and thigh, and also the labia and scrotum, that are apt to suffer from this affection.

The appearances found after death from erysipelas are similar in many respects to those found in cases of typhus fever.

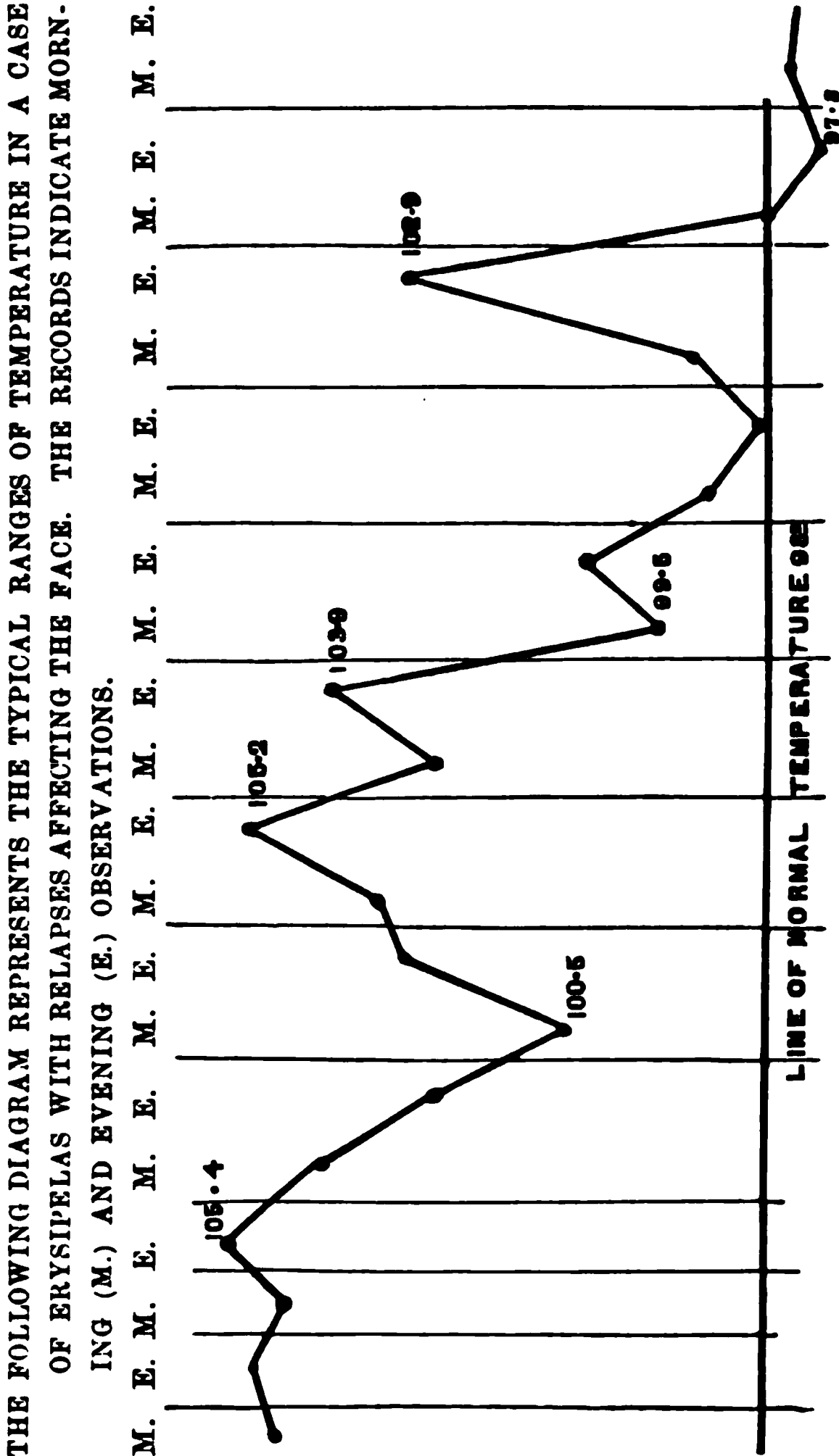
**Symptoms.**—The symptoms of erysipelas arise out of the fever and local affection, and appear of various degrees of intensity.

In acute sthenic cases the erysipelatous inflammation is preceded and accompanied by fever; and the attack may be sudden, or ushered in by rigors, irregular flushings, muscular pains, accelerated pulse, white tongue, nausea, vomiting, and deranged bowels. Sore throat is an early and constant accompaniment. These symptoms, when they do exist, last for some hours—perhaps till the end of the second night or beginning of the third day—when the fever becomes continued, the tongue brown and dry, and shortly afterwards the cutaneous inflammation appears, but without any remission of the fever. The inflammation generally appears at the seat of any injury to the skin, such as a wound, and is most intense there. By some, indeed (Trousseau, for example), it is held that erysipelas always originates from some external injury or irritation, which may be very slight. But this character erysipelas has only in common with other eruptive diseases, as Mr. Paget has described in his admirable Address on Surgery, delivered to the British Medical Association at their meeting in London, in August, 1862. He noticed that, “having cut a boy for stone, the boy became very ill three days afterwards, and seemed in danger of his life; but soon a vivid red eruption appeared at and about the wound. This was measles, earliest and most intense at the seat of injury, just as erysipelas might have been. Thence it extended, and ran its ordinary course, and did no harm.” Mr. Paget states that he has seen similar events with scarlet fever, the eruption commencing in an injured and inflamed knee. Dr. William Budd records similar events in a case of small-pox, in which the eruption first appeared and was most intense over a bruise on the nates. The argument from such facts is, “that the local determination of erysipelas, and of all other allied diseases, after operations, is no proof of their local origin or local nature.”

**Diagnosis.**—The diagnosis of erysipelas is, in general, easy. For a few hours, perhaps, if a joint be attacked, it may be mistaken for acute rheumatism; or if a surface be attacked, it may be confounded for a short time with erythema, but the intumescence and spread of the disease quickly enable us to rectify the error.

Frank has pointed out a symptom which he considers diagnostic—namely, that whenever a patient has exhibited, for twenty-four

or forty-eight hours, an intense febrile movement, attended with *pain, swelling and tenderness of the lymphatic glands of the neck*, he does not hesitate to announce the approaching development of erysipelas; and in no case has the diagnosis been invalidated by the result.



The course of the fever in erysipelas is very similar to that of measles; but the advance of the fever to its height continues longer, and the epoch for the commencement of the defervescence vacillates between the *fourth* and the *eighth* days. The defervescence, as a rule, is rapid, the normal heat being attained, or nearly so, in from twelve to thirty-six hours. Frequently, however, the case is not terminated therewith. New relapses may take place, and the course

of the disease may be prolonged through two or even three weeks. These relapses are severally of short duration; but they come on again and again, and are ushered in by a smaller or larger increase of heat, and they are connected with a renewed spread of the cutaneous affection; and it is only after the eruption has ceased that complete and definite defervescence ensues. Very sudden changes of temperature are characteristic— $4^{\circ}$  or  $5^{\circ}$  Fahr. in twenty-four hours, or a fall of  $7^{\circ}$  or  $8^{\circ}$ , commencing immediately on the appearance of the characteristic redness (COMPTON). This erratic and protracted form of erysipelas is most frequently met with in the aged, associated with gouty or rheumatic states of the system, as well as with albuminuria or renal disease. The *local* symptoms vary according to the part affected, the mode of termination of the inflammation, and also according to the character and duration of the fever.

When erysipelatous inflammation affects the face, it may begin either in the skin or in the subjacent areolar tissue. If the areolar tissue be primarily affected, the face at the inflamed part becomes swollen, but the skin suffers no discoloration for some hours, so that it is impossible to distinguish it from an ordinary attack of swelled face. At length, however, the skin inflames, and the part is now red, hot, and painful, as well as swollen, and the disease is fully formed. At the commencement of erysipelas of the face the attack is usually partial, and perhaps limited to the bridge of the nose, to one ear, to the lower eyelids, or to one cheek; but in severe cases it gradually extends, often involving the whole of the integuments of the face, head, and neck; so that at the end of three or four days those parts present a strangely swollen, disfigured, and even in some instances, hideous appearance, scarcely a feature being discernible. The nostril, moreover, is imperforate from internal swelling, so that the patient is obliged to breathe with his mouth open, while the inflammation may extend to the auditory passages, and render him completely deaf. Extension of the inflammation to the membranes of the brain sometimes takes place, while the external inflammation continues. This untoward event is followed by delirium and coma. But delirium frequently supervenes in the course of erysipelatous attacks, independently of any metastasis or extension of the disease to the membranes of the brain. It commences with wandering of the mind at night, similar to that which is observed in fever. Utterance is given for the most part to low, muttering, and rambling expressions, which rarely assume a noisy character, but which in fatal cases terminate by coma. When the patient has been of dissipated habits, or is otherwise of a dilapidated constitution, then the delirium resembles that of *delirium tremens*, not due to inflammation of the brain, but in consequence of an altered condition of the blood and of the nervous system (BARCLAY).

On the fourth, sixth, eighth, or some later day, the bright red color of the skin changes to a deeper hue; the serum effused is absorbed, desquamation takes place, and the skin gradually returns to its natural color. It is not unusual, however, for abscesses to form, particularly on the eyelids or cheeks, and which, being opened quickly, heal, and hardly retard the convalescence of the patient.



and young children, are most frequently the subjects of gangrenous erysipelas; and it is not uncommon in hospitals, during the prevalence especially of malignant epidemics of erysipelas.

**Cause.**—The mystery which hangs over the origin of disease poisons does so, in a remarkable degree, over erysipelas; for this disease is often epidemic, and appears to be very constantly present in communities, and especially in large towns.

The predisposing conditions are age, mechanical or chemical injuries, as blows, punctured wounds, and incised wounds generally, bites of insects, or burns; also certain articles of diet, as mussels or periwinkles; and many diseases likewise, as dropsy with renal disease, typhus fever, and others of a debilitating kind. The effects of age in predisposing to this disease are considerable. New-born children, for instance, are occasionally subject to it, but from that period to adult age it is seldom witnessed. The period of life most subject to acute attacks is from twenty to forty; and to frequent asthenic attacks from forty to old age. Both sexes suffer in nearly equal proportions.

**Propagation of the Disease.**—The spread of erysipelas has been so frequently observed, both in the sick-room and in the wards of hospitals, that no doubt can exist of this disease being communicable by impalpable emanations. In the year 1760 erysipelas spread so extensively through the wards of St. Thomas's Hospital, in London, that it was believed the plague was in the hospital. Dr. Baillie described it as spreading also in St. George's Hospital, London; and Dr. Cullen, in the Hospital at Edinburgh. It has been found to spread extensively on board ship; and Drs. Wells, Watson, and others, have given several remarkable instances of its spreading in families. Dr. Steele writes, in his excellent *Annual Report on Guy's Hospital* for 1863, that "for some years past it has been customary to place patients suffering from erysipelalous wounds in these (the medical) wards, in order to diminish as much as possible the risk of extending infection in surgical wards, as well as to promote recovery in the patients themselves. It happened, however, that in one of the wards of the new hospital into which a patient suffering from erysipelas was placed in the course of the past year, *five* persons suffering from other complaints were attacked with the disease; and although none of the cases were attended with fatal consequences, the occurrence is sufficient to point out the danger which must be occasionally apprehended." Dr. Maclachlan, on the other hand, has never seen the disease propagated by contagion or infection in the infirmary of Chelsea Hospital; and he is disposed to think that the disease is less contagious or infectious when occurring in persons of advanced life than at other periods.

That it is communicable by some palpable virus, was shown by Dr. Willan, who says, that if a person be inoculated with the fluid contained in the phlyctenæ or vesicles of a genuine erysipelas, a red, painful, diffused swelling and inflammation analogous to erysipelas is produced. The danger, however, attending this experiment has not allowed it to be repeated.

Erysipelas also spreads by *fomites*. In hospitals, wards are occa-



sixty than to people between fifteen and sixty years of age (*Registrar-General's Fifth Report*, p. 456).

**Treatment.**—Broussais states that when he served with the French armies in Italy, he has seen erysipelas allowed to run its natural course, and the result was, that it made immensely rapid progress, and ended either in suppuration, in gangrene, or in fatal visceral inflammation.

Erysipelas, in the opinion of some, is a disease of simple inflammation, and consequently ought to be treated by general and local bleeding; while, on the contrary, others contend that it is a specific inflammation; and long experience has shown that bleeding is often injurious, while a tonic mode of treatment is much more uniformly successful.

There are very few physicians, from the days of Hippocrates to the present time, who have not bled patients in erysipelas, and consequently this experiment has been made on a large scale; still, many of the warmest advocates of bleeding admit that the operation is occasionally followed by unpleasant consequences. Indeed, the treatment by bleeding has been often followed by so many unfavorable results, that many physicians, the most intelligent of the profession, affirm that, according to their experience, the practice is not only unfavorable but highly injurious. Andral is reported to have said, "In erysipelas with delirium, bleeding pales the skin, but the disease continues; the cellular tissue remains gorged, and death follows. We open the body, but find nothing." Cruveilhier says, "*des erysipèles rentrés*" is a consequence of unusual or too abundant bleeding, and he considers the question of bleeding in this disease to have been "*depuis longtemps jugée.*" Blache and Chomel likewise say that "Experience has proved that general bleeding has no other effect than to blanch the eruption, without notably abridging its duration." In this country, Drs. Fordyce, Wells, Pearson, Heberden, and Willan, all give their testimony to the frequent ill effects of bleeding in this disease; and, in consequence, they, for the most part, recommend a tonic treatment.

It is therefore to be recollected that bleeding will not cure the *erysipelatous* inflammation, in the way that it produces a salutary effect on an idiopathic inflammation of the lungs, occurring in an otherwise healthy person. It is also to be borne in mind that as a rule, bleeding is not borne well by persons suffering from erysipelas; and it is necessary to be ever mindful of the fact that people of a certain class in populous towns cannot bear bleeding so well as those who pass their life in the country. For instance, a brewer's drayman in London, accustomed to rejoice in the beverage which he delivers to his customers, would sink suddenly under the influence of a bleeding: when if double or even treble the amount of blood were abstracted from a countryman suffering from erysipelas of a sthenic form, but heretofore in good health, it would produce but little effect, and that probably for good. Bleeding, as a rule, is only indicated in the young, the healthy, and the vigorous; and it must equally be avoided entirely in the aged and in broken-down cachectic patients.





A line of circumvallation is to be painted round the erysipelatous part, so as completely to inclose it. The nitrate of silver should either be employed in the solid stick, or as proposed by Higginbottom, in solution of eight scruples of the nitrate with twelve drops of nitric acid in a fluid ounce of water. Dr. Wood has practised with success, and recommends the use of tincture of iodine.

[Dr. Addinell Hewson reports (*Trans. of the College of Physicians of Philadelphia*, 1867), that in extensive trials of the local use of the sulphite of soda, in solution of ten grains to one ounce of water, he has had most decided and prompt results. He has never seen it fail to arrest the progress of the disease when thoroughly applied before the deep planes of cellular tissue had been invaded. Lint soaked in the solution is thoroughly applied not only over the affected surface, but to a considerable distance beyond it, and covered with oiled silk to hinder evaporation. In the first twenty-four hours the discolored surface is sensibly bleached, and in forty-eight hours all traces of the disease have disappeared. He had thus cured twenty-seven cases of erysipelas, seven of which were idiopathic.]

Long and deep incisions into the inflamed textures are sometimes demanded. This is more especially the case if there be tension of fibrous tissue, such as the subcutaneous fasciæ; and erysipelas of the head is frequently greatly alleviated by repeated innumerable minute punctures, made by the point of a lancet all over the parts of the face and scalp which are affected.

### THE PLAGUE.

LATIN, *Pestilentia*; FRENCH, *Peste*; GERMAN, *Pest*; ITALIAN, *Peste*.

**Definition.**—*A specific malignant fever which has prevailed at different times and places epidemically; attended with an eruption of a complex nature, composed of buboes or swellings of the inguinal or other lymphatic glands, and occasionally with carbuncles, pustules, spots, and petechiæ of various colors, and distributed in different parts of the body.*

**Pathology and History.**—Modern medicine restricts the term “plague” to a disease of dreadful severity, and of a peculiar character, which appears to have been first recognized in Egypt and in the neighboring countries. It is impossible to determine the time when the plague first appeared in Egypt. The remotest period to which we can distinctly trace it is when spreading into other countries, as the plague of Constantinople, which broke out in 544, when Justinian was emperor. This is the first time that the disease, from its course and symptoms, can with certainty be recognized as the plague of more modern times. The symptoms were shivering and fever, at first so slight as to alarm neither the physician nor the patient; but the same day, the next day, or the day after, there appeared swellings of the parotid, axillary, or inguinal glands, with carbuncles, and sometimes gangrene; and from the more usually diseased state of the glands it was called “*pestis inguinaria*.”

The disease from that period has prevailed at short intervals in



ity often infiltrated with serum, and occasionally with a trifling effusion of black blood. The substance of the brain was generally less consistent than in health, and sprinkled with more bloody spots than usual. The bronchial membrane appeared sensibly inflamed, although during life the patient had presented no catarrhal symptoms. The pericardium frequently contained a reddish serosity. The serous membrane covering the heart and pericardium was often extensively affected with petechiæ. The heart, distended with blood, was almost always enlarged from a third to a half greater than its natural size, its tissues being often pale and sometimes softened.

In acute cases the stomach and small intestines were softened, and presented similar petechial appearances.

The liver was almost always larger than natural, and loaded with blood, while petechial spots were often seen at its surface. The gall-bladder was the seat of petechiæ, and in two cases blood was effused into the submucous areolar tissue.

The spleen was always twice its natural size, or even more, but was rarely the seat of hemorrhagic effusion. It was softened, and deep in color.

The kidneys were often found immersed in a hemorrhagic effusion into the surrounding tissue. They were loaded with blood, and the pelvis filled with clots. The ureters occasionally contained blood, and sometimes the lumbar glands were so enlarged as to press upon them, and to account for the suppression of urine. The bladder occasionally presented petechiæ, and occasionally the urine was mixed with blood.

Every dissection showed that buboes, wherever seated, always resulted from enlarged lymphatic ganglia, varying in size from an almond to a goose's egg. The least altered were hard and injected. In a more advanced stage some of these glands were without any change of color, and others again as richly colored as lees of wine, and either wholly or partially softened or putrescent. Sometimes these glands became agglomerated, forming masses which weighed two pounds or more, and around these agglomerations a hemorrhagic effusion extended into the areolar tissue. The cervical glands often became so enlarged as to form a sort of chaplet, united with those of the axilla and of the mediastinum. The axillary glands, again, communicated with the cervical, and with those which surrounded the bronchi. Those in the groin connected themselves in the same manner with those of the abdomen, and these might be traced without interruption through the crural arch into the pelvis and along the vertebral column. It was especially among these latter that sanguineous effusion was found in the subperitoneal tissue. The mesenteric glands were often so numerous that the whole of the mesentery seemed covered with them, but they seldom exceeded an almond in size.

In the Mediterranean cities, where plague epidemics have prevailed, it is of importance pathologically to remember that epidemics of "anthrax," "carbuncle," "phlegmon," "boil," or "pustule," are not uncommon. The disease usually shows itself in the form



destroyed the eye. Clot Bey, however, observed they never formed on the scalp, the palms of the hands, or on the soles of the feet.

There are three different varieties of carbuncle, and all commence in the same way, or by a small red pimple, which increases, and in the centre of which a vesicle forms, containing first a yellow and afterwards a blackish serum. In the most benign the vesicle bursts, and dries up in three or four days from its first formation, the epidermis alone having been infected. The second variety involves the whole thickness of the skin, as well as portions of the cellular tissue, which is moderately tumefied, and surrounded by a dark-red areola. The gangrene in this form is circumscribed, and there results an eschar from one to two inches in diameter, which is detached by suppuration, leaving an ulcer with a sharp perpendicular edge. In the severe forms the redness and tumefaction cover a large space, and the gangrene rapidly involves the skin, the cellular tissue, and sometimes even the bones. It has been observed that the malignity of the carbuncle is in the direct ratio of the severity of the disease, but the mere existence of carbuncle is not of unfavorable augury. Their number is very various, sometimes only one, at others ten or twelve. When there are several, they often form in succession. These tumors are often very painful; and Aubert mentions one, seated on the back of an Arab soldier, four inches in diameter.

Petechiæ are observed in some seasons and not in others. They present different shades of color, according to the intensity of the disease—rose color, violet color, or black. Aubert considered their appearance an almost certain sign of death. The duration of the disease is from a few hours to fifteen, twenty, thirty, or even more days.

**Diagnosis.**—Clot Bey says the diseases which most resemble the plague are typhus fever, severe forms of paludal fever, apoplexy, dysentery, parotitis, and scrofulous or syphilitic affections associated with febrile symptoms of a typhoid type.

**Cause.**—The plague, and the specific poison which it generates, seem to have a very limited geographical range. Clot Bey, indeed, considers it to be endemic along the whole of the eastern and southern coasts of the Mediterranean, the principal centres of propagation being Egypt, Syria, and Constantinople. But most authors are agreed that Egypt is the great focus of the plague, whence it may be propagated under circumstances of overcrowding, filth, dampness, and organic decomposition. It seems determined also that the disease is often circumscribed within a very small space of country. Volney states that in Egypt the plague never commences in the interior, but always appears first on the coast at Alexandria, passes from Alexandria to Rosetta, and from Rosetta to Cairo.

All that we can safely affirm of the poison of the plague is, that it is at all times endemic in Egypt, along with the cognate diseases of "carbuncle," "anthrax," or "boil," already referred to, and every five or six years it becomes epidemic. It also appears to be, to a certain extent, influenced by season, not spreading in any very



with the loss of the army generally. In the English army only one in forty-eight of the military died of the plague, while one-half of the medical officers died. Some few persons also have ventured voluntarily to inoculate themselves with plague-matter, and these have, with hardly an exception, fallen victims to their rash experiments.

Dr. Russel states that at Aleppo he met with twenty-eight cases of re-infection, or 1 in 157; and Clot Bey states that he and his colleagues saw many individuals perish of plague in 1834–35 who had formerly survived an attack of the disease.

**Treatment.**—In the treatment of the plague neither the practice of the French nor English medical officers serving in Egypt has led to any happy result; and it is to be regretted that recent experience has not in any degree advanced the successful treatment of the plague. “In the beginning of the epidemic,” says Clot Bey, “when the morbid cause acts with a rapidity so great that some hours are sufficient to compromise the life of the patient, every treatment, even the most energetic, is powerless to arrest the course of the disease. When, however, the intensity of the disease abates, we may hope for the recovery of the patient.” Looking, however, to the pathology of the disease, and regarding it as a form of malignant *typhus fever*, the principles of general treatment ought to be similar to those laid down in the account of that disease.

We have no sufficient evidence to prove that plague may be carried beyond those geographical limits where it or the cognate diseases already noticed are epidemic. Quarantine establishments to prevent the transmission of such epidemic diseases are now therefore unwarrantable nuisances, and vexatious interruptions to mercantile enterprise. I am informed by a medical friend who visited Malta in 1861, that a curious instance of the wavering nature of opinion regarding the efficacy of quarantine was afforded during the last Benghazi plague. The Maltese—the most sensitive people formerly on the subject—absolutely declined to put any quarantine on arrivals from Benghazi, and trade went on as usual until the Austrians intimated that, unless Malta put Benghazi in quarantine, Trieste would put Malta in quarantine; and the poor merchants were obliged to submit. There is little risk of plague now, because of the great improvements which have taken place; and it is to be hoped that as sanitary measures are developed the barbarism of quarantine will entirely disappear; except, perhaps, where the intelligence of the people does not go beyond that of the Governor of Eupatoria, who requested that the allied armies of France and England might go into quarantine when they landed in the Crimea in 1854! (KINGLAKE.)

## SECTION II.—THE CONTINUED FEVERS.

Fevers have been classified according to various theories; and much has been written on the subject. In the previous section those diseases have been described in which an eruption on the skin especially challenges attention, and with the appearance and development





the "anatomical sign," described by Louis. The first opinion was that this "anatomical sign" was an incidental occurrence; or, that its occurrence was in some way connected with locality, the cases of fever being everywhere considered identical. A second opinion, however, soon began to gain ground, especially when the intestinal lesion was not found by the most careful observers in some cases where it was intentionally looked for (as in the epidemic of Toulon in 1829-30). A belief now, therefore, began to gain ground that there were in fact *two diseases*, which were indifferently named *typhus* and *typhoid fever*—that one prevailed only at Paris, and the other in England, in Germany, and elsewhere, being also sometimes more or less mixed up with the Parisian fever, as measles may be with scarlet fever. Louis subsequently (1841) adopted this view.

In 1835 the "Académie de Médecine" formally proposed the question, "What are the analogies and the differences between the *typhus* and *typhoid* fevers?" The question excited considerable interest in France, but less so in England, where a strong bias has always prevailed towards a belief in the doctrine of a single fever—a belief entertained and taught by the most eminent observers and teachers of the day. But dissenters arose. Scotch, English, and American physicians, practically familiar with the fevers of their countries, began to visit Paris to study fever there; and they were not long in learning to recognize the chief point of difference between the two fevers. Gerhard and Pennock, of Philadelphia, in a systematic treatise, were the first to indicate (1836) these differences, it having been already determined by Jackson and Gerhard that the fever described by Louis under the name of *typhoid fever* existed in America, and presented there the same assemblage and development of symptoms, and the same post-mortem lesions, as the Parisian fever.

In 1836 M. Lombard, of Geneva, after visiting London, Edinburgh and Dublin, ultimately came to the conclusion that two different fevers had been confounded together; and Drs. Staberoh, of Berlin, and Kennedy, of Dublin, professed the same belief through the same medium—the *Dublin Journal*. During this year (1836), also, Dr. A. P. Stewart commenced his observations in the Glasgow Fever Hospital, where he continued his inquiry for two years. His attention was first especially directed to the study of fever by Dr. Peebles, who, during a long residence in Rome, had observed the maculæ of typhus in the contagious fever of Italy, and who first showed the difference between the characteristic eruption of fever and the cutaneous affection to which the name of "*petechiæ*" is given (*Edin. Med. and Surg. Journal*, 1835). He pointed out this eruption to Dr. Perry (then Physician of the Glasgow Fever Hospital), "and who," Dr. Stewart states, "was the first to maintain the complete difference of the two eruptions—namely, those of *typhus* and *typhoid fever*." Dr. Stewart subsequently went to Paris and examined the fever there. The result was a complete recognition of the existence of two fevers, and of their differences—an account of which he published in the *Edinburgh Medical and Surgical Journal* for 1840, p. 289.

In 1839 Enoch Hale published an account of the fever of Massa-



post-mortem appearances from the other cases (in which the "anatomical sign" referred to was present) as to render it impossible to suppose that they were cases of the same disease? Or,—contrary to the opinion of Louis,—Were the symptoms of the two sets of cases so similar as to lead to the belief that the presence or absence of the intestinal lesion (the "anatomical sign") was a matter of little consequence?

On comparing these two groups of cases, Dr. Jenner found that while the symptoms and post-mortem appearances of the 23 cases were exactly the same as those described by Louis, the symptoms, course, and post-mortem appearances of the remaining 43 cases were entirely different—so different, indeed, as to render their separation from the other cases a matter of absolute necessity, if accuracy was to be maintained in the description of these diseases, or certainty arrived at in their treatment.

Causation, as a ground of distinction between the two fevers, is a condition upon which much stress has been laid by Dr. Jenner, and subsequently by Dr. Murchison. Dr. Jenner was the first to argue that the material media by which the two fevers are propagated are specific and different from each other, according as they are generated by the bodies of those affected with the one or the other form of fever. This argument he based upon the circumstance, that because *certain local foci* sent *typhoid* cases to the hospitals, and *certain other local foci* sent *typhus* cases there, he inferred that different specific causes existed in each focus. Dr. Murchison has also clearly stated the evidence of many other observers, which goes to prove that the two fevers have no community of origin (*Continued Fevers of Great Britain*, p. 588).

[Dr. Southey Warter (*St. Bartholomew's Hospital Reports*, 1866) considers the thermometric differences between typhus and typhoid fever so great as to completely settle any doubts as to their being separate diseases.]

This brief history of the progress of our knowledge regarding typhus and typhoid fevers has been mainly condensed from an erudite and most interesting monograph on "The Diagnosis of Fevers," by Dr. Parkes, which appeared in the *Medico-Chirurgical Review* for July, 1851—a contribution of not less importance to science than the original investigations of those whose labors it records; for it connected the scattered observations together, and showed at once the practical value of the discovery that had been so gradually made—tending, as it did, to bring conviction to the minds of those not fully conversant with the literature of the subject, and with what had actually been achieved in different parts of the world. To Dr. Parkes, the clear, elaborate, and careful analysis he made was a labor of love—justly believing, as he does, that no subject is so important as an accuracy of diagnosis. It is the foundation of therapeutics; and he who clearly indicates how a disease can be recognized is fellow-laborer to him who points out how the disease may be cured or prevented.



## ENTERIC FEVER—Syn., TYPHOID FEVER.

LATIN, *Febris enterica*; FRENCH, *Fièvre typhoïde*; GERMAN, *Abdominal-typhus*—Syn., *Ileo-typhus*; ITALIAN, *Tifo enterico*.

**Definition.**—A continued fever associated with an eruption on the skin of rose-colored spots, chiefly on the abdomen, appearing generally from the eighth to the twelfth day, occurring in crops, each spot continuing visible about three days. Languor and feebleness are prominent from the first, attended by headache, abdominal pains, and (early) by spontaneous diarrhœa. With the advance of the disease the diarrhœa increases, the discharges being for the most part liquid, copious, of a bright yellow color, devoid of mucus, occasionally containing altered blood; in reaction alkaline, and containing a large proportion of soluble salts and some albumen. The disease may terminate favorably by a gradual restoration to health during the fourth week. The average duration of the fever is about twenty-three days. Death in the majority of fatal cases occurs towards the end of the third week. There are symptoms also associated with the characteristic lesion of this form of fever—namely, fulness, resonance, and tenderness of the abdomen; more or less tympanitis, with entire effacement of the natural lineaments of the belly; gurgling in the iliac fossæ; increased splenic dulness. The specific lesions are enlargement of the mesenteric glands, with deposit in the glands of Peyer and in the minute solitary glands of the small intestine.

**Pathology and Symptoms.**—Typhoid fever begins gradually—often, indeed, so very insidiously that its commencement is not always able to be fixed.

This form of continued fever is described under a great variety of names, by various writers, such as *typhus mitior*; *nervous fever*; *abdominal typhus*; *common continued fever*; *entero-mesenteric fever*; *dolinententeritis*; *follicular enteritis*; *bilious fever*.

The fever may be ushered in with rigors, chilliness, or profuse diarrhœa; and amongst the early symptoms, the most characteristic are the abdominal pains and diarrhœa, which continue to increase. [There is early muscular debility, shown by the staggering walk, and, subsequently, by dorsal decubitus.] The countenance indicates anxiety, [and has a distinctive besotted expression]; the mind continues clear, [but intelligence soon becomes weakened, and questions often have to be repeated before understood and answered; this partly depends on dulness of hearing, with ringing in the ears, which are very constant]; and delirium, when present, is generally active. [Frontal headache is a constant initial symptom, and often insomnia.] The patients are vivacious, and disposed to leave their beds. The conjunctivæ are pale, the pupils dilated, the cheeks somewhat flushed, and slight, though sometimes excessive, epistaxis not seldom occurs, at repeated intervals, during the first week. The belly enlarges, as in mesenteric disease, and is resonant on percussion. Gurgling on firm pressure may commonly be detected in the right iliac fossa, and there is often tenderness in the same situation, [with pain, on pressure, around the umbilicus.] From the seventh



sometimes it is violent and loud, and occasionally hysterical. The face is swollen and dusky, and the skin over the malar bones of a livid red, and there is often general capillary sluggishness of the surface of the body. The pulse is soft, rapid, and, sometimes, irregular; the duration and intensity of the first sound of the heart will, in many cases, be found lessened, and may become quite extinct.

Towards the end of the second week, or the beginning of the third, there is either a gradual, though marked abatement in the symptoms, or they suddenly and quickly worsen. If the attack is to end in recovery, the temperature of the body lessens, chiefly in the morning, and the skin becomes moist and soft; the tongue cleans, and the buccal secretions return; the expression of the face begins to look more natural, and the pulse is slower and steadier. Or, if the duration of the disease is to be prolonged, a decided increase in the severity of the symptoms will take place, and new ones will be added. The tongue becomes drier, browner, fissured, and trembling; sometimes it is of a bright red color and smooth, as if covered with a coat of varnish. The mouth and teeth are crusted with dark sordes; there is great difficulty, or even inability, to protrude the tongue, or to swallow, which may be due to paralysis of the muscles of deglutition, but more often to the half-dried mucosities gathered about the base of the tongue. The nostrils become blocked with dried mucus or blood, and the breathing has a peculiar whistling sound. The pulse is quick and irregular, reaching 120 beats or more; meteorism is excessive, and diarrhœa is profuse and often, the stools passing involuntarily; there is retention or suppression of urine, or, though rarely, incontinence; and hemorrhages may happen from the nose, bowels, or vagina. Bronchitis becomes more general and intense, and pneumonic complications set in; at this time uræmic coma may come on, and petechiæ appear. The heat of the body is acrid; a peculiar odor is, sometimes, exhaled, said by some to be like that of mice; sloughs are common on those parts of the body which have been exposed to pressure, as over the sacrum, heels, scapulæ, trochanters, &c., or gangrene may attack blistered surfaces, or leech-bites, or parts of the skin where sinapisms have been applied. Sometimes spontaneous sphacelation takes place; Dr. Grisolle has seen gangrene of the integuments of the thigh, scrotum, foot, and lower lip come on in the course of typhoid fever without any obvious cause.

The duration of convalescence is generally proportionate to the sharpness of the attack; when this has been severe and protracted, and the prostration great, strength is slowly gained, and recovery is very gradual. Emaciation is often excessive at the beginning of convalescence. Painful œdema of the lower extremities, rarely extending to the upper limbs, and face, and loss of the hair of the head are frequent. In many cases the hearing remains dull for some time, particularly where there has been a purulent discharge from the meatus. Convalescence may be suddenly arrested by symptoms of gastric disorder, the digestion becoming difficult, the skin hot, and the pulse quick, arising from some irregularity of diet; they commonly abate after a day or two. Abscesses, eschars, erysipelas, and successive crops of boils on the trunk and extremities, often lengthen convalescence. A persistent frequency of pulse may last for some weeks. Autophagic vertigo is not uncommon after a protracted attack, and particularly where the patient has been imperfectly nourished. Paralysis, dependent on deficient innervation, both of sensation and motion, may complicate convalescence, causing blindness, deafness, paresis of the lower extremities, or loss of power in the sphincters of the rectum or bladder. Softening of the cornea has been noticed.





tions. Derangements of the sensory functions are of three kinds: (a) Exaltation of function,—hyperæsthesia and spontaneous pains; (b) Perversion of function,—abnormal sensation of cold or heat, prickings and formillation in the extremities and along the spine; much more rare than the first variety, and, usually, when present, associated with it. (c) Diminution or abolition of function,—the several degrees of analgesia and anæsthesia, cutaneous and muscular.

Hyperæsthesia may be limited to the skin, or to the muscles, or both may be affected. Cutaneous hyperæsthesia may extend over a considerable portion of the body. Its site is often the skin of the abdomen, or of the extremities, or conjointly, the lightest pressure, or the merest touch being intolerable; when it is of less degree it may be provoked by gently pinching a fold of integument, or passing the finger over the internal face of the tibia, about the malleoli, or the condyles of the femur. Next in frequency we have increased sensibility over the spinous processes, sometimes reaching from the atlas to the sacrum, and sometimes limited to a single apophysis, and induced by pressure. There may be severe aching pains in the muscles, generally of the lower limbs; or rachialgia, radiating to different parts of the body, and increased by any movement; violent pains in the chest and waist; and neuralgia, which is commonly symmetrical. Both cutaneous and muscular hyperæsthesia may be chiefly complained of toward evening, when the body-temperature rises. Its progress is generally regularly ascending, and it disappears in inverse order.

Dr. Robert Law, of Dublin, thus graphically describes the spinal symptoms which were observed in the Famine Fever of 1848: "The most common and loudest complaints of our patients was an aggravation of what the subject of fever generally describes as pains in the bones, but which really means pains in the course of the spinal nerves. While in former fevers this complaint was seldom more than that of a contused or bruised feel, or of such a sense of discomfort or fidgetty restlessness as the French so significantly express by the term *malaise*, here the individual in many cases seemed to suffer as intensely as in the severest cases of acute rheumatism. These pains were more or less general in different patients. In some they affected the back of the head and neck; in some they only ran down the legs; while in others they spread themselves through the whole body, and embracing the sides, imparted the sensation of painful constriction. The nape of the neck and across the loins were the points to which the patients most frequently referred their pain." (*Dublin Quarterly Journal of Medical Science*, Nov., 1849.)

Derangements of the motor functions are: numbness of the extremities, paraplegia, partial paralysis of the respiratory muscles, retention of urine, paralysis of the sphincters, spasm, or irregular contraction of the muscles of respiration or of the extremities, and muscular rigidity, particularly of the muscles of the neck and limbs. The special group of nervous symptoms originating in the medulla oblongata, are: extreme breathlessness, not due to any morbid condition of the organs of respiration, spasm of the pharynx and larynx, convulsive cough, aphonia, glossoplegia, spasmodic or rhythmic contraction of the sterno-cleido-mastoid and trapezius muscles, and paralysis of the pharynx.

When a patient with typhoid fever has died after or during the occurrence of the spinal symptoms just described, no appreciable material lesion has been found in the cord or its membranes. In a very limited number of cases can these symptoms be referred with any strictness to a congestion of the cord (FRITZ).

When the spinal symptoms of typhoid fever have been very marked and



all the symptoms, including the eruption, not unfrequently supervene. He must not be betrayed into the belief that danger is past, if, towards the eighth or tenth day, the little headache that prevailed may pass away, and the other febrile phenomena just mentioned may subside. It is on record that events such as these have led to the belief that convalescence from a mere "febricula" had been established, leading to the discharge of the unfortunate patient from hospital. His vocation, if a soldier, would then compel him to undertake severe duties during the actual height of a severe disease, made more dangerous and perhaps fatal by such a mistake.

Another symptom, often very painful, is *meteorism*, or the accumulation of air in the large intestine. This is present in a greater or less degree in one-half of the cases, and when considerable it always marks a grave affection, and one generally fatal. On the contrary, the abdominal muscles are, in a few cases, tense and strongly contracted. It is, however, the experience of all physicians that there is no condition so low, and no symptoms so severe, from which the patient may not recover; and, on the other hand, there is no case of this form of fever so slight that it is to be considered free from danger. The prognosis must therefore be cautious, because perforation of the intestine may follow the mildest case, and death from peritonitis ensue.

The symptoms of typhoid fever cannot be said to be fully expressed till the characteristic eruption has appeared.

The Eruption consists of the so-called *rose spots* peculiar to *typhoid* fever, the "*taches rosées lenticulaires*" of Louis. They begin to appear from the sixth or seventh to the twelfth or fourteenth day of the disease, very rarely later, and still more rarely at an earlier period than the sixth day. A very delicate scarlet tint of the whole skin, closely resembling the skin of a person soon after leaving a hot bath, sometimes precedes, by a day or two, the characteristic eruption of typhoid fever; and this is important to remember, because it may be mistaken for the rash of scarlet fever, especially if sore throat is present. The eruption consists of slightly elevated papulæ or pimples; but, to detect their elevation, the finger must be passed very delicately over the surface of the skin, because, although pimples, they are not hard, like the first day's eruption of small-pox. Their apices are neither acuminate nor flat, but invariably lens-shaped or rounded, and the bases gradually pass into the level of the surrounding cuticle. No trace of vesication can be detected on their apices. They are circular, and of a bright rose color, the color fading insensibly into the natural hue of the skin around. Their margin is never well defined. *They disappear completely on pressure*, resuming their characteristic appearances as soon as the pressure is removed. These characters they preserve from their first appearance to their last trace. They leave behind no pit, scar, or stain. They vary in size, but their usual diameter is nearly 2 lines, but varying from 1 to 2½. The ordinary duration of each papula is about two days, but its existence varies from two to six days, and fresh ones generally make their appearance every day or two after the first appearance of eruption, and they continue



fected by pressure. They form and disappear slowly, last for some time, fading one day and deepening the next; few in number, from four to ten, they are found on the abdomen, upper part of thighs, base of thorax, and rarely on the extremities. The time of their appearance is variable, being sometimes as early as the first week. They would seem to be more common in certain epidemics than in others. Some observers have regarded them as a species of ecchymosis, and the first stage of petechiæ, and connected with the blood lesion; but one of many objections to this view is that when present it is constantly in light cases. Though, it is believed, peculiar to typhoid fever, their rarity takes from their diagnostic importance.]

**Typhoid Fever in Children.**—It has been now clearly established that typhoid fever is by no means an unfrequent disease amongst children, and has been often described under the name of "*Infantile remittent fever*." [It occurs sometimes epidemically. This has been noticed at the Children's Hospital at Paris, and Dr. Rilliet saw an epidemic typhoid fever in a small village near Geneva (Switzerland), which attacked children only.] Boys seem to be more liable to attack than girls. [Of 121 cases recorded by Rilliet and Barthez, 81 were boys and 31 were girls; and of 121 observed by Taupin, 86 were boys and 31 girls.] It is most frequent between six and eleven years of age; and from five to nine seems the period of greatest liability. Its occurrence is rare during the first years of life. Nevertheless, it is on record at the following very early ages: namely, between two and three months; three months; six months; seven, ten, and thirteen months (WUNDERLICH, HENNIG, FRIEDRICH, RILLIET). The author of a very interesting Review on the typhoid fever of children, in the *British and Foreign Medico-Chirurgical Review* for July 1858, p. 161, mentions, in his own experience, the occurrence of typhoid fever in a girl one year and seven months old; and also in a boy two years of age.

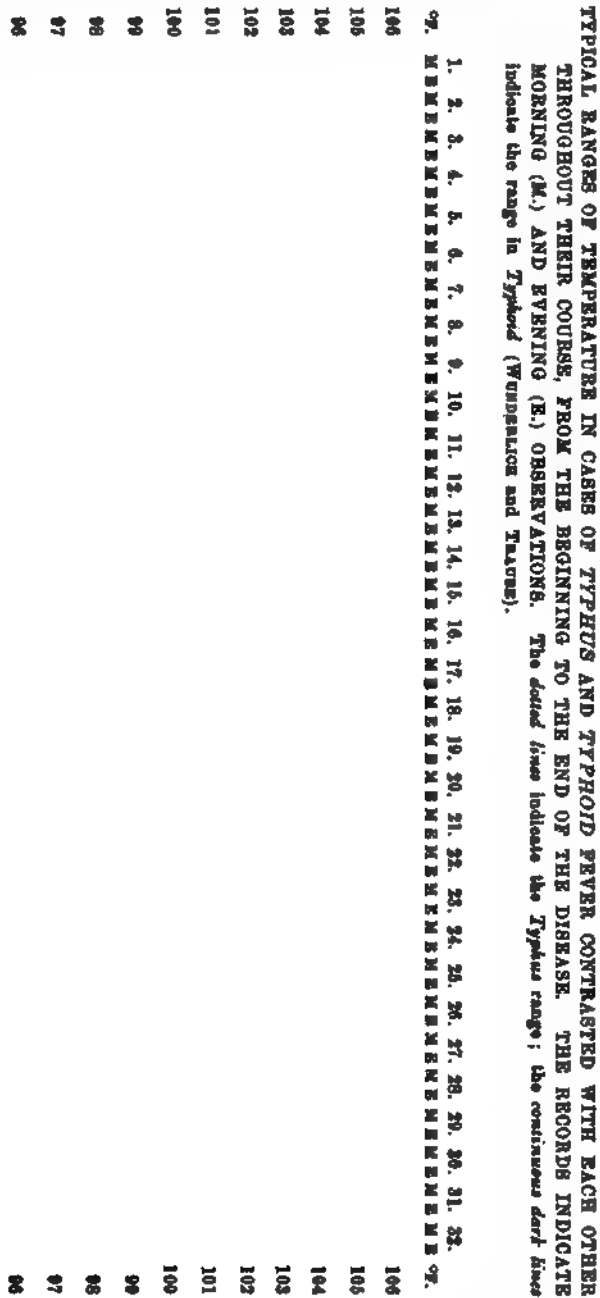
The chief symptoms of typhoid fever in the child are,—[epistaxis,] splenic enlargement, diarrhœa, meteorism, gurgling in the course of the colon; associated with pyrexia, quickened respiration, bronchial catarrh; delirium, somnolency. [The tongue, though dry, is rarely fissured or cracked; retention of urine is infrequent; vomiting, particularly at the outset, is common. Peritoneal perforation, intestinal hemorrhage, and gangrene of the intestine, seldom happen; otitis often occurs.] The eruption already described, and sudamina, are nearly constant in children after five years of age. The rose-colored spots are especially frequent on the back and the extremities, so that, if the abdomen and chest only are examined, their presence may often not be apparent.

**The Temperature during Typhoid or Intestinal Fever.**—Wunderlich has given a summary of results derived from the observation of 700 cases of typhoid fever, investigated thermometrically (*Arch. der Heilk.*, vol. ii, 1861, p. 433; also, *Edin. Med. Journal*, Nov., 1862, p. 465).

The course of the disease is typical, and the type is characteristic; and when irregular cases occur, irregularity may in general be traced to a special cause. The mode of accession is pretty nearly the



A retardation of recovery until at least the fourth week is to be anticipated when in the second week the morning temperature is



above 103° and the evening above 104.5°; when the exacerbations occur early in the forenoon and remain after midnight; and, lastly,





complished under great excitement of vascular reaction, renewed hyperæmia, sloughing, softening, and final cicatrization.

Cases intermediate in severity between the mild and severe cases just described are not unfrequently met with. Many of them, although they show a course more or less irregular, nevertheless follow a pretty clearly defined type as to variations of temperature, and are capable of clinical recognition. There are, still considerable evening exacerbations during the second week, yet with a tendency to abatements in the mornings. During the third week great vacillations between morning and evening temperature continue, and sometimes also between single days. During the fourth or fifth week the normal temperature is reached in the morning; but it is only in the fifth or sixth week that the temperature becomes permanently normal—the evening temperature showing a complete freedom from fever—so that the beginning of convalescence can only be established with certainty by the use of the thermometer.

In the majority of cases of typhoid fever, severe as well as mild, a peculiar periodicity of weeks and half weeks cannot be mistaken. Each week shows a distinct character, which cannot be overlooked in a graphic representation. On the first and last days of each week changes generally take place which are either temporary changes or continue till the fever subsides.

**Duration of Attack and the Mode of Recovery**, or the transition into the feverless state, is peculiar and characteristic of enteric fever. With rare exceptions, the defervescence is a remittent one. The great vacillations between morning and evening recur for a longer or shorter interval. For weeks the evening temperature may amount to 104° Fahr. or more, whilst in the morning the patient is quite free from fever. At the same time the transition into the feverless condition may follow different courses. The remissions may either become longer and longer—the morning temperature decreasing and the evening remaining stationary; or after some time the remission may become shorter and shorter—the evening temperature, together with the morning temperature, gradually descending. Again, the differences between the morning and the evening temperatures may remain nearly the same, while a relative decrease takes place at both periods; or the fever shows a sudden transition into the remissions with low temperatures—changes which generally correspond with the commencement of weeks. The period of development of the disease occupies two weeks, or a week and a half in slight cases; in severe cases it may occupy two and a half to three weeks. The initial stage (that is, the period when the growth of material in Peyer's patches takes place) lasts about half a week. The removal or elimination of the growth may take place in a week; but the process may extend over several weeks. In mild cases the disease continues at its height for only a week or a week and a half,—rarely for two weeks; so that the whole duration of a mild case of typhoid fever extends from eleven to eighteen days. The period of convalescence occupies from one to two weeks. The whole disease, therefore, in mild cases may be



It occurred in a female twenty-five years of age. Diarrhœa was considerable; and blood was largely passed in fluid stools the night before the seventeenth day of the fever. On the morning of the seventeenth day the temperature was as low as  $93^{\circ}$  Fahr., rising in the evening to about  $101^{\circ}$  Fahr. After the eighteenth day diarrhœa ceased; but the differences between the morning and evening temperatures continued to be very great; and it was not till the twenty-sixth day that these differences began to grow less and less.

The approach of death is indicated by a permanent or persistent elevation of temperature in the morning (as high as  $106^{\circ}$ ); by a sudden rise to  $108^{\circ}$ , or even higher; and more seldom to a depression below  $93^{\circ}$ .

**Condition of the Urine in Typhoid Fever.**—It is not till the third or fourth day of the fever that the urine assumes any special characters. It is peculiar in the following respects:

I. As to normal constituents: (1.) *The water* is greatly diminished, generally about one-half or even to one-fourth or one-sixth. This lessening of the water is most marked during the first week; it then begins to increase gradually during the second and third weeks; and at the end of the fourth week, in favorable cases, it has reached its normal standard. (2.) *The whole amount* of the urine does not seem to stand in any close relation to the febrile heat; but when the temperature begins to fall permanently, the urine increases at once, or very soon after. (3.) *The specific gravity* of the urine is high in almost all cases where the urine is scanty; and at convalescence the specific gravity diminishes, sometimes before the amount of water increases; i. e., at convalescence the lessening of the solids of the urine is often prior to the increase in the water. (4.) *The urea*, as a rule, seems to be augmented, during the febrile period, above the physiological standard proper to the individual; and it sinks again below this standard during convalescence. The amount of increase varies: Vogel has noted 78 grammes, or 1200 grains, in 24 hours; while Parkes has noted 57 grammes, or 880 grains, in that time. In most of the cases observed by Dr. Parkes the average increase has been about one-fifth above the physiological standard proper to the individual; and the augmentation is most marked in the first week, when the water and the chloride of sodium are at the lowest point; and if the fever be continued beyond the third or fourth week, the urea keeps up in amount. The relation of urea to temperature is yet uncertain. (5.) *The chloride of sodium* is diminished (indefinitely); the cause of the diminution being in part due to the lessened ingress of this substance on account of spare diet: or due to the elimination of large quantities of it with the stools or the sweat. (6.) *The uric acid* is uniformly increased in amount; and it is relatively greater than that of the urea. It is often doubled in amount; and the increase progresses up to the fourteenth day, when it is at its greatest. It then diminishes to the twenty-first or twenty-eighth day; and during convalescence falls below the normal amount. Spontaneous deposits of urates occur very frequently; and when there is no such deposit it may be brought about by a drop of acid; but as yet the



amount of fever and *formation of them with elimination*. The greater the excretion in typhoid fever the better; and as long as 500 to 700 grains of urea in men, or 300 to 500 in women, are being passed in each twenty-four hours, the progress so far is favorable. But whenever, while the fever continues, the urea falls much below these amounts, we may anticipate a low typhoid condition, or some local inflammation, as pleurisy, which may relieve the blood for a time from some of the effete products, but which at the same time may kill the patient.

The existence of slight albuminuria or hæmaturia is not of itself unfavorable; but if either be in large amount, or if there be exfoliation of epithelium or renal cylinders present in the urine, retention of urea and its consequences may be expected.

**Morbid Anatomy of the Lesions in Typhoid Fever, with special reference to the Phenomena and Progress of the Disease.**—The abdominal complications of typhoid fever, as they are sometimes called, are mainly due to lesions of the solitary and aggregate glands of Peyer, and to enlargement of the mesenteric lymphatic glands. This lesion in the ileum is especially recognized as the “anatomical sign” of enteric or typhoid fever. It is necessary to remember, however, in connection with the age of typhoid fever patients, that the solitary vesicles and the aggregate glands of Peyer are known to be most fully developed and most active in youth, up to the age of early manhood; after that time they begin to disappear, and are obviously less active in the adult after thirty years of age. Structure and function seem to be alike impaired by age, till at length, after forty or forty-five years, traces only of their existence are apparent, or they have altogether disappeared. The gland substance (whose structure has been so well described by Dr. Allen Thomson, Kölliker, and Boehm) no longer exists; and the places where the patches of Peyer once were may be detected only after careful examination,—a mark of varied form and character being all that indicates the place of the patch. There is therefore a good anatomical reason why typhoid lesions are rarely found after fifty years of age, and seldom after forty. Dr. Jenner records only three cases beyond fifty—namely, one at fifty-one and two at fifty-five. Dr. Wood has observed one case at fifty-five years of age. Dr. Murchison notes two cases above sixty-five, and refers to five other cases between sixty and seventy-five, related by MM. Lombard and Gendron. Dr. Wilks refers to the case of a woman aged seventy, of very doubtful history (*Path. Society*, 1861). These exceptional cases are explicable when it is known that the existence and functional activity of these glands are sometimes prolonged for an indefinite term of years beyond the usual period of their existence. On the other hand, it is in childhood and early life that these glands are most obvious, and their functional activity the greatest; and therefore it is extremely significant to find that “more than one-half of the cases of typhoid fever occur between fifteen and twenty-five years of age; and in very early life the proportion of cases of typhoid would be greater were it not that many children laboring under this disease are described as cases of ‘*Infantile Remittent Fever*’” (MURCHISON).



intense irritation of the mucous membrane—*catarrhal* and *gastric* symptoms—prevail; and the mucous membrane generally is swollen and turgid, especially the villi of the intestines, which are particularly distinct, imbedded in a thick layer of dirty-yellow gelatinous mucus. Although these conditions seem to involve the whole of the mucous membrane in the first instance, yet they soon begin to be more expressed towards the lower end of the small gut than in any other part. The time at which this increase of cell-growth commences in these glands is not yet well defined in relation to the day of the fever. It seems certain, however, that it occurs within the first week; but it may be later. A case is described by Dr. Sankey, in the first volume of the *Pathological Society's Transactions*, in which dissection showed the growth of the glands as early as the fifth day. The bulging of the patch and the extent of intumescence vary considerably in different patches; and simultaneously with these conditions the mesenteric glands begin to increase in size. They, too, are supplied with an increased quantity of blood, and the increased tissue of the gland becomes unusually soft and elastic.

III. *A subsidence of the general congestion, and of the generally turgid state of the mucous membrane, takes place after the gland-growth has been fully developed.* Nevertheless, the growth continues actively, and progresses rapidly till the patches of Peyer become so thick as to be elevated three or four lines above the surface of the mucous membrane. A beautiful vascular halo encircles them, stopping short at their margins; and a contracted border surrounds the margin of the patch, which gives it a sessile fungiform aspect, with an umbilicated-like depression on its surface. Growth is now confined within narrow limits, pressing on the muscular coat below and the mucous coat above. The patches assume various aspects as to color; and, when vascular, they have an appearance which has acquired for them the description of being like “fleshy lumps;” their tawny gray color showing through the peritoneum of the gut. Varicose vessels abound in the vicinity,—a fact of some importance in connection with the formation of thrombi, and which may lead to hepatic or pulmonary embolism. The specific gravity of the mucous tissue of Peyer's patches is obviously changed by such increased growth, ranging from 1.032 to 1.044.

IV. *Softening of the contents of the tumid gland-cells* seems to be the next event in the series, and which would appear to be preliminary to one or other of the following results, namely:

V. *Conditions under which the softened contents of the glands begin to be eliminated.* This elimination seems to take place in one or other, or in each of the three following ways; conveying out of the body by the intestinal discharges abundance of morbid material, presumed, with great probability, to contain the specific virus of the fever:

1. *Elimination without ulceration—simply by the rupture of the hitherto closed gland-vesicles.* This is the usual and natural way in which the vesicles of Peyer's patches become open follicles in the course of their normal physiological existence. For many reasons I am induced to believe that this is the natural, the most common,





mass appears to be about the ninth or tenth day (MURCHISON); and the softening which precedes ulceration is associated with a return of the violent congestion to the small intestines, when the veins especially are filled with dark-colored viscid blood. The outbreak of the ulcers is always characterized by an aggravation of the original symptoms, after it may have been sanguinely supposed that convalescence had decidedly taken place. But in such deceptive convalescence the abnormal temperature is maintained, showing with absolute certainty that the fever is not at an end. The ulceration therefore is usually denoted (*a*) by a re-accession of the febrile phenomena, with or without diarrhoea; (*b*) by abdominal pains and tenderness. Judging from post-mortem examinations, the ulceration seems to commence at the lowermost patches in the glands nearest to the cœcum, and the ileo-cœcal valve is often implicated in the destruction. The ulcers vary in number and in extent; and although there is a tendency to perforation of the gut in fatal cases, yet actual perforation is not common, and peritonitis may supervene without perforation having actually taken place. Various statements have been made concerning the tendency of typhoid ulcers to perforate the gut, and the frequent association of this lesion with peritonitis. Perforation is said to be rare in the northern parts of Europe (HUSS); but, from the records of Drs. Murchison and Bristowe, it appears to be a more frequent mode of fatal termination than has been commonly supposed in this country. Of fifty-five fatal cases, perforation occurred in eight (LOUIS); of fifteen fatal cases, perforation occurred in three (MURCHISON); of sixty-three fatal cases, perforation occurred in twelve (London Fever Hospital Records); of fifty-two fatal cases, perforation occurred in fifteen (BRISTOWE).

From these data it appears that perforation occurs in about one in five fatal cases; and it generally takes place through the ileum near the valve. Post-mortem examination often discloses vigorous attempts on the part of neighboring structures to limit by union and adhesion the results of perforation, obviously indicating, in practice, the necessity of absolute rest throughout the disease.

The characters which distinguish the ulcers of typhoid fever from other ulcers of the intestines may be stated as follows: (1.) They have their seat in the lower third of the small intestine, their number and size increasing towards the ileo-cœcal valve. (2.) They vary in diameter from a line to an inch and a half; but a number of ulcers may unite to form a mass of ulceration several inches in extent. Such extensive masses of ulceration occur close to the cœcum. (3.) Their form is elliptical, circular, or irregular—elliptical when they correspond to an entire Peyer's patch, circular when they correspond to a solitary gland, and irregular when they correspond to a portion of a Peyer's patch, or when several ulcers unite to form one. (4.) Elliptical ulcers are always opposite to the attachment of the mesentery. (5.) The ulcers never form a zone encircling the gut, as may sometimes be seen in the case of the tuberculous ulcer, but their long diameter corresponds to its longitudinal axis. (6.) Their margin is formed by a well-defined fringe of mucous membrane, detached from



days slightly, on the sixteenth day to the extent of  $4^{\circ}$  below  $98^{\circ}$ , on the seventeenth day to the extent of  $5^{\circ}$ , on the twentieth day to the extent of  $2^{\circ}$ . These falls of temperature were all traceable to the influence of repeated hemorrhages from the bowels. The occurrence of hemorrhage is always a most alarming symptom, and is most frequent during the third and fourth weeks of the disease. It varies in amount from a mere stain to a large quantity of blood, sometimes discharged in clots, and generally of a red color, in consequence not only of the rapidity with which it is passed out, but also, as Dr. Parkes has shown, in consequence of the alkaline reaction of the contents of the intestine. It may cause immediate death by syncope; or, by reducing the temperature and strength of the patient, he may sink exhausted, unable to cope with the disease. Whenever, therefore, blood appears in a case of typhoid fever, it is certain that the lesions of Peyer's patches are severe.

In addition to these three modes of elimination of the new growth from the intestinal glands, there are reasons for believing that it may be occasionally *reabsorbed*; unless such cases where resolution, independently of ulceration, commencing about the tenth day, may not be explained by the first method of elimination I have described.

VI. The mucous membrane of the intestines having existed for several weeks in a state of irritation which has been described, and the catarrh being more or less excessive, *an atrophic condition of the intestine at last supervenes*. The mucous tubes become wasted, irregular in form and size, sometimes separated by an interstitial growth of a granular nature, their bulbous ends disappear, and the whole substance of the gut becomes so thin that it resembles a portion of thin paper rather than intestine.

The mesenteric glands are invariably enlarged. They begin to enlarge at the very commencement of the disease, and sometimes attain a very large size, and their stages of congestion, of swelling, and of subsidence, go on simultaneously with the similar changes in Peyer's patches.

The spleen is usually greatly enlarged, varying from five or six to fourteen ounces, with a specific gravity varying from 1052 to 1059. Its Malpighian sacculi (glandular) are also intumescent.

Pulmonary lesions occur (1) as infiltrations, or (2) as the consolidation of pneumonia, or (3) as portions of lung which have become carnified.

In the first-mentioned form of lesion the growth seems to commence in the terminal air-vesicles, ultimately assuming the form of a miliary deposit, with a semi-transparent gelatinous appearance. It is the irritation set up by this sudden growth which generally gives rise to pneumonic consolidation. Softening and friability of the pulmonary texture is thus a very constant post-mortem state in protracted cases of typhoid fever. Such lesions usually supervene during the later period of the fever, and when the ulcerations of the intestines are extremely spread (Huss). In this respect only it differs from the consolidation of the lung to be described in typhus fever. This lesion has been also termed *non-granular consolidation*, dependent for its origin and development on a specific cause; and



which may either complicate the progress of the case or come on subsequently to the fever. It is the development of tubercle. Usually when recovery takes place from *typhoid* fever it is complete; but in some cases, especially where there is hereditary predisposition, an impetus or tendency seems to be given to the development of tubercles in the lungs. If the physical signs of bronchitis continue beyond the thirtieth day, or fourth week, combined with hurried and difficult breathing, and with the signs and symptoms of great irritation of the lungs, then there are good grounds for suspecting that the deposition of tubercle has commenced in the lungs. Dr. Stokes gives two sets of cases in which this deposit takes place. In one set a great quantity of tuberculous matter seems to be formed during the existence of the fever; and although, sometimes, such an occurrence may not have been suspected, yet the expectoration of pulmonary calculi, at periods of different duration after the convalescence, furnish strong proofs that such a lesion had taken place. In other cases, again, the cure may be effected through absorption, or by suppuration of the minute tuberculous points over the mucous surface of the bronchia. A doubtful convalescence, a quick pulse, and a hectic state, suggest such a state of things, especially when combined with persistent bronchitis.

Erysipelas, phlebitis, parotitis, and such like local inflammations, are not uncommon in typhoid fever. Such lesions may be excited by cold simply; but the absorption into the blood of putrid substances, from the ulcerating patches of Peyer or other diseased parts, may be usually, and probably correctly, considered to be the cause of most of the secondary inflammations already noticed to occur in typhoid, enteric, or intestinal fever. Dr. Parkes considers it probable, however, that deficient urinary excretion may have a share in their production (Parkes *On the Urine*, p. 254).

Such are the more obvious secondary affections which may develop themselves during the progress of *typhoid* or *enteric fever*, and the derangements which these give rise to constitute new phenomena in its course. In some severe cases, however, the fever may destroy the patient in a few days, without leaving a trace of organic lesion in any part of the body.

These secondary affections just noticed all arise after the fever has existed some time; and it appears now to be pretty well established that the intestinal lesion at least is a special growth, which, in cases of recovery, follows first a progressive or developmental course, and afterwards retrogrades; just as in variola we first observe the development and maturation of the pustule, and subsequently its disappearance. The same may be said of the other local lesions in typhoid fever, although the existence of a special growth is not yet so fully established in the case of the thoracic and cerebral lesions, or in the parenchymatous, as compared with the mucous structures of the intestine; still, it is believed by some that an action more or less analogous to that which occurs in the *glands of Peyer* and the *minute solitary closed vesicles of the ileum*, occurs also in all the secondary lesions of typhoid fever in other parts (Dr. Stokes). Specific characters of the elements composing the growth cannot be



symptoms which typhoid fever has in common with typhus fever, cholera, small-pox, dysentery, scarlet fever, diphtheria, ichorrhæmia. The intensity of the fever (measured by the thermometer) is generally great in those cases, and the fatal event occurs either at a very early period of the fever, associated with cerebral congestion, or it may occur later, when it may be supposed that the danger is past. This is sometimes termed the secondary poisoning of the blood (septicæmia), and is most likely due to the ulcerated intestines, with the bowels perhaps on the verge of perforation. The pulse becomes rapid and small; cold, clammy sweats appear; and the body begins, even in life, to exhale a putrid odor. In cases where the blood is so gravely implicated, gas has been observed to become developed during life, and has been detected in the veins at the root of the neck for some minutes before death (CLOSS, FRANK, and JEFFREY MARSTON in *Med. Times*, Feb. 7, 1857).

2. By implication of excretory organs at an early period—for example, the kidney, as denoted by albuminuria, or by bloody urine—conditions which tend to aggravate the blood poisoning.

3. By congestions of important organs—for example, the lungs and the brain, in consequence of poisoned blood; and which congestions are still further brought about by the circulation, in the bloodvessels, of putrid juices, or of the substance of fibrinous debris of clots in a granular condition, having formed as plugs in the varicose veins surrounding the sloughs and ulcers of the intestines.

4. By hemorrhage from the bowels during the separation of the gland-sloughs.

5. By exhaustion from profuse diarrhœa in cases where the catarrh of the mucous membrane has been excessive.

6. By peritonitis, with or without perforation of the intestines. There are two periods in the course of the fever when perforation is apt to take place. The first period is during the separation of the sloughs, about the end of the second and throughout the third week. The second period is during protracted convalescence, with atrophy of the intestine already described, and when the ulcers are in a weak atonic state, the result of intense protracted fever and profuse catarrh.

7. By peritonitis subsequent on suppuration of the large mesenteric glands, and rupture of their inclosing capsule (JENNER); or from the bursting of softened new growth from the spleen into the peritoneum (ROBERTSON, JENNER); or from ulceration of the gall-bladder. The average mortality among cases of typhoid fever appears to be about 1 in  $5\frac{1}{2}$  to 1 in 6. It is considerably less in autumn than in spring; and is least of all in winter. It tends to be greater among males than females; and the average age of fatal cases appears to be about 23.5. The mortality increases to a small extent as life advances. The disease in certain places seems never to be absent, and is invariably most prevalent during autumn, at the time that diarrhœa is most common; and it has been observed to be especially prevalent in seasons remarkable for their high temperature (MURCHISON).

[**Prognosis.**—The prognosis in typhoid fever should be always very





of an epidemic of typhoid fever, and if, on careful examination, no local disease can be found, there will be good reason to suspect the presence of the disorder, even though the so-called distinctive symptoms are wanting. The absence of the prodromic symptoms special to the exanthemata, should prevent its being mistaken for them. The visceral inflammations of old persons are often latent, and accompanied with extreme debility; but a regard to the time of life, and careful physical exploration, will generally make the case clear. Granular meningitis in children, may be mistaken for typhoid fever. Besides the absence of the special symptoms of typhoid fever, as well as of the bronchial complication, in granular meningitis there are commonly constipation, frequent vomiting, retraction of the abdomen instead of tympany; and active delirium is an early symptom, followed by deep coma. The facial expression of the two disorders is totally different. Acute phthisis in some respects often resembles typhoid fever; but the great and increasing difficulty of respiration, the presence and site of the moist crepitant rhonchi, general lessened resonance of the chest, the peculiar hue of the surface, and, indeed, the absence of the really distinctive phenomena of typhoid fever, should hinder us from confounding two disorders, which really have but few common elements, and sufficiently distinguishing ones, to prevent an erroneous diagnosis.

The late Dr. Southey Warter has published some valuable thermometrical observations respecting the differential diagnosis of typhoid fever and typhus (*St. Bartholomew's Hospital Reports*, 1866). In typhus the rise of temperature pretty generally follows that of the pulse; but in typhoid fever he did not find this to be the case. While the pulse rarely is more than 120 before the 8th day, and this number is uncommon, the temperature reaches from 102.4° to 104.3° Fahr. In typhoid fever the highest afternoon temperatures are reached about the 4th day; the highest in typhus usually on the 7th, sometimes on the 8th, and rarely as late as the 11th or 12th days. Both in typhus and typhoid fever there is a singular tendency of the body-heat to rise every alternate day, especially in the odd days; in typhus rises are common, on the 13th and 16th days; in typhoid fever, on the 9th, 11th, and 15th.]

**Origin and Propagation of Typhoid Fever.**—It is now about thirty years since M. Bretonneau related to the French Academy of Medicine a series of cases in which the communication of this disease from person to person, and its modes of propagation in this way, were so evident as to admit of no reasonable doubt. Nevertheless, the conclusion arrived at has not been generally accepted by the profession, so that the communicability of typhoid fever has not met with general belief.

[Though, as a general rule, the contagious principle of typhoid fever is not very active, it is incontestable that the disease is communicable, and, as Dr. Trousseau remarks, the number of those who deny it lessens daily. Most of the French physicians living in the rural districts have always advocated its contagiousness, whilst those of Paris for a long time denied it. Dr. Nathan Smith, as far back as 1824, maintained that it was as contagious as small-pox or measles. In July, 1829, Dr. Bretonneau transmitted to the French Academy of Medicine a communication, in which he asserted the contagiousness of dothientérie, as it prevailed in the country (*Archives Gén. de Méd.*, t. xxi, p. 57). Chomel, in his *Leçons de Clinique Médicale*, published in 1834, inclined to this opinion, though, as he acknowledges, it was contrary to the general sentiment. Gendron, of



fever. Twenty-nine others carried the seeds of the fever with them, and were laid up by it at their own homes. Of these twenty-nine, it was ascertained that *as many as eight communicated the disorder to persons who were engaged in attendance upon them.*

In further illustration of the doctrine of communicability, Dr. Budd cites, with minute details, numerous examples in the village of "North Tawton," in which typhoid fever having once appeared in a household, it extended itself to one or more members of the family before it finally died away. During the prevalence of the fever in this village, it also so happened that three persons left the place after they had become diseased, and each of the three persons communicated the same disease to one or more of the persons by whom they were surrounded in the new neighborhood whither they went. While two of these men remained in the village of "North Tawton," they both lodged in a court having a single and a common privy in it, and next door to a house where typhoid fever was. In due course of time and events both took the disease. The third man was a friend who came to see one of the two men already sick. He assisted to raise his sick friend in bed, and while so employed was quite overpowered by the smell from the sick man's body. The sense of this pestilent smell harassed him for days. He felt very unwell from that time; and on the *tenth* day from the date of the event just noticed, he was seized with shivering, followed by the complete expression of an attack of typhoid fever, which was of long duration; and before he became convalescent two of his children were laid up with the same disease, as well as a brother, who lived at some distance, but who repeatedly visited him during his illness. Except in the houses of these men no fever existed in that part of the country. Further, most interesting and conclusive examples are given in Dr. Budd's admirable papers published in the *Lancet* of 9th July, 1859, which prove beyond question that typhoid (intestinal) fever is a most readily communicable disease. The facility for propagation, however, seems to be modified under particular circumstances of season, place, and habits of life; in other words, the propagation of the disease requires some special conditions, which may be said of the whole class of communicable diseases.

2. The disease having once occurred, the patient is protected from a second attack. The specific nature of the disease is thus also established, for the fever not only propagates itself, but propagates no other kind of fever—one case following another with the same constancy of specific type that small-pox follows small-pox or measles succeeds to measles.

3. There seems also to be a definite period in which the poison is latent after being communicated—a period of incubation, during which a definite interval elapses before the development of the fever begins. This period, according to Dr. Budd's experience, seems to be from about a week to ten or fourteen days. *The living human body, therefore, is the soil in which this specific poison breeds and multiplies; and that most specific of processes which constitutes the fever itself is the process by which the multiplication is effected.*



decomposition and dissemination by the air and water supply. Diarrhœa was common. Badly constructed sinks within a few yards of the camp were the rule, and not unfrequently no sinks were used, but the environs of the camp were converted into a general latrine. Privy odor was soon developed, and the drinking-water contaminated. When sufficient time had elapsed, typhoid fever declared itself amongst those who had not completed the seasoning process, or those who had acquired a special susceptibility to the poison." (*United States Sanitary Commission's Memoirs of the War of the Rebellion*, p. 200.)]

**Preventive Measures, or Measures for Checking the Spread of Typhoid Fever.**—The measures about to be specified have been made public through the writings of Dr. William Budd on this subject; and, *provided they are thoroughly and efficiently carried out*, it is believed that the recurrence of typhoid fever may be entirely prevented.

To enable us to judge of the extent of the infection to be destroyed, there are two elements to be taken into account,—*First*, The amount and duration of the intestinal discharge in each case; and, *Second*, The number of cases actually occurring. With regard to the first, Louis has found that the average duration of the alvine flux in cases of typhoid fever is *fifteen* days in mild cases and *twenty-six* days in severe cases. With regard to the second point—namely, the number of cases occurring—the Reports of the Registrar-General show that at least 100,000 to 150,000 cases of typhoid fever occur annually in England alone. In other words, "*every year in England more than 100,000 human intestines, diseased in the way already described, continue each, for the space of a fortnight or thereabouts, to discharge upon the ground floods of liquid charged with matters on which the specific poison of a communicable disease has set its most specific mark*" (BUDD).

The measures recommended for preventing the spread of this fever are founded on the power of chemical agents to destroy absolutely the material which contains or carries the specific virus of such communicable diseases. Assuming it, therefore, to be certain that the intestinal discharges in typhoid fever are the media of propagating the disease, it is no less certain that, by SUBJECTING THE DISCHARGES ON THEIR ISSUE FROM THE BODY TO THE ACTION OF POWERFUL DECOMPOSING CHEMICAL AGENTS, THEY MAY BE ENTIRELY DESTROYED OR DEPRIVED OF THEIR SPECIFIC VIRUS. He suggests the following details of procedure:

1. All discharges from the fever patient should be received on their issue from the body into vessels containing a concentrated solution of chloride of zinc.

2. Two ounces of a caustic solution of chloride of zinc should be put in the night-stool on each occasion before it is used by the fever patient.

3. All tainted bed or body linen should, immediately on its removal, be placed in water strongly impregnated with the same agent.

4. The water-closet should be flooded several times a day with a strong solution of chloride of zinc; and some chloride of lime should be also placed there to serve as a source of chlorine in the gaseous form.



puerperal fever, and cholera, yet there does not seem to be sufficient evidence to show that any of these causes can *produce* a disease which is of so specific a nature as to be maintained and propagated by a specific poison generated in the body alone. Undoubtedly, the state of ill-health induced by the decomposing material of night-soil and the like, does produce *a state of the system favorable to the development*, not alone of typhoid fever, but of many other specific diseases, such as cholera, dysentery, yellow fever, and the like. This predisposition to such diseases seems to be exactly analogous to the preparation of a soil for seed. Dr. Carpenter, also, long ago, showed physiologically what observation has since confirmed—namely, that decaying animal material, especially night-soil, seems to be for some poisons (*e. g.*, cholera and yellow fever) great centres or foci, where the specific germs or poisons are able to multiply; and for the propagation of which “foulness of medium is indispensable.” An interesting question for inquiry is thus opened up as to whether the germs of typhoid fever, cholera, and the like, could be made experimentally to grow or increase upon or about organic matter, just as the germs or spores of many fungi are induced to grow in collections of manure (DR. LANKESTER). In such collections on the earth’s surface there is reason to believe that germs of diseases like cholera and typhoid fever, and yellow fever, may find a resting-place—that thus they are always extant somewhere—although it may be only now and then, when season and other conditions conspire, that they display their full power as epidemic diseases. As such, they seem to occur every now and then as “mysterious cycles,” the existence of which we admit, but do not understand. The experiments of Dr. Barker on cesspool air prove that long inhalation of an atmosphere charged with the gases evolved from decomposing organic matter is capable of producing a series of symptoms of the following character—namely, increased heat of the skin, thirst, irregular and feeble muscular contractions, and diarrhœa. These symptoms continue so long as the person is exposed to the influence of the foul air; but when the cause is removed, there is no continuance of symptoms, no recurrence nor remittency, but a tendency to recovery. No communicable disorder is induced. The poison of the foul air acts for the time as chloroform might act, and so soon as removed, recovery progresses.

The history of typhoid fever, whose leading features have been described in the previous pages, is wholly inexplicable upon the “pythogenetic theory” of Dr. Murchison. On the contrary, it is emphatically the history of a specific disease generating a specific poison, and propagating itself by it:

“*Mutatis mutandis*,” writes Dr. Budd, “it is the history of *small-pox*, it is the history of *scarlet fever*, it is the history of malignant cholera. In all these specific contagions we meet with these same alternations of slumber and activity; of widespread prevalence in one place, while other places hard by remain free; and finally, with the same successive invasion of neighboring places, in such wise that the reigning disorder—be it *small-pox*, measles, scarlet fever, intestinal fever, or malignant cholera—





Professor Jones further remarks: "If the contagious nature of typhoid fever be admitted, might not the relative greater proportion of persons liable to the disease in the country and villages, where it more seldom prevails, as well as the intimate relations and associations, and constant visiting amongst the entire population, and especially the collection together at stated periods in one or more houses of worship of a large proportion of the inhabitants, account for the apparent more extended action of the fever after its introduction into small towns and villages, without the necessity of resorting to the hypothesis of the propagation of the disease by the emanations of the cesspools? However, if the entire lesion of typhoid fever be the characteristic manifestation of the disease, corresponding to the eruption of small-pox, we must admit that the theory of Drs. Budd, Simon and Aitkin, has much plausibility, and even probability. According to this view the discharges from the bowels in typhoid fever might be regarded in the same light, as far as their contagious nature was concerned, as the matter formed upon the surface, and cast off in the form of scabs in small-pox. The admission of this theory does not at all overthrow the assertion that animal putrefaction does not generate under any circumstances a contagious fever, for in the case of the excrements voided in typhoid fever, the poison is the result of the actions going on in the living body, and is not the product of the decomposing excrements" (p. 603).]

**Treatment of Enteric or Typhoid Fever.**—The chief indications of treatment are to reduce temperature and subdue vascular excitement, if these be in excess; to restrain and moderate, but not to suppress or check, the diarrhoea; to stimulate the nervous system when necessary; to obtain a free action of the kidneys; and to influence the elimination of the morbid growth from the intestinal glands.

To accomplish the first of these indications the use of *digitalis* has been especially recommended by Wunderlich. He considers that it decidedly mitigates the febrile symptoms which are present in severe cases at the time when the ulcers begin to heal, and which often impede or prevent recovery. He advocates its use in the severe forms of the fever only, especially at a time when most danger is to be apprehended from the violence of the fever in the second week, when the evening temperature is at its highest (105° to 108° Fahr.), and when the remissions in the morning are slight; when the pulse is frequent, 110 to 120, or more. In mild cases it is superfluous. He finds that in the form of *infusion* it is easily absorbed by the intestines of patients suffering from fever; and, if given in a suitable dose, has most marked effects in subduing the rate of pulsation, and in reducing animal heat. Large doses of the infusion should be given without interruption until the full effect has been obtained,—

An infusion of fifteen or twenty grains of *digitalis* in eight or ten ounces of boiling distilled water may be consumed in twenty-four hours by adult patients.

It acts more rapidly on animal temperature than on the heart. For the first few days after its use the decrease of temperature is rather slight, but may afterwards become considerable; and after it



small and repeated doses, [or prepared chalk, with or without bismuth.] This latter remedy retards the peristaltic action of the intestines, and lessens the secretion from the mucous membrane. The dose must be regulated so as to avoid vomiting; and the feeling of nausea which is apt to follow the first dose soon disappears with continued use.

Dr. Murchison, on the other hand, agrees with the late Dr. Todd, who writes as follows: "Restrain diarrhœa and hemorrhage in typhoid fever, and when you have fairly locked up the bowels, keep them so. Patients will go for four or six days, or even longer, without suffering inconvenience from this state of constipation." Dr. Huss and Dr. Murchison speak highly of the benefits to be derived from the mineral acids—hydrochloric and sulphuric\* especially. From fifteen to thirty minims of the dilute acids may be given every three or four hours; and with each dose Dr. Murchison recommends half a grain of quinine, as in the following prescription for an adult:

R. Acid. Sulph. dil., vel Acid. Hydrochlor. dil., ℥xx. ad. xxx.  
 Quiniæ Sulph., gr. ½ ad. gr. j.  
 Syrup. Aurantii, ʒss.  
 Aquæ Carui, ad. ʒj.  
 Fiat haustus, 3â vel 4â, horâ sumendus (MURCHISON).

He is of opinion that if there be more than two motions in the twenty-four hours, with marked prostration, that astringents should be had recourse to. A starch enema, containing from ten to twenty drops of laudanum, should be administered towards evening, and recourse may also be had to the following draught:

R. Acid. Sulph. Aromat., ℥xxx.  
 Liq. Opii. Sedativ. (Battley), ℥ij.  
 Aq. Menth. Pip., ʒj; *misce*.  
 Fiat haustus, 4tâ vel 6tâ, quaque horâ sumendus (MURCHISON).

If the mineral acids are not tolerated by the stomach, acetate of lead is worthy of trial, in doses of two or three grains in solution every four or six hours, with or without an eighth of a grain of morphia (MURCHISON).

Alum dissolved in gum, to the amount of twenty-four grains in a day, which may be increased to one drachm, is best given in the

---

\* [With a view of ascertaining any curative influence that might be exerted by sulphuric acid in continued fever, Dr. Irving W. Lyon instituted a comparison in the rate of mortality in the male fever ward at the Bellevue Hospital, New York, in cases treated with and without the acid, during periods of six months. He left out all cases where death took place within forty-eight hours after admission. From January 1st to July 1st, 1863, 70 cases of fever were treated without sulphuric acid, with 14 deaths, or 20 per centum. From July 1st, 1863, to January 1st, 1864, 78 cases were treated with sulphuric acid; there were 8 deaths, or 10.25 per centum. The general treatment in both series was essentially the same. The apparent reduction of mortality was one-half (*Am. Med. Times*, February, 1864). Magnus Huss, of Stockholm, very strongly recommends dilute phosphoric acid, in hourly doses of about ten minims.—EDITOR.]



late Dr. Anthony Todd Thomson used to give it; and, from the observation of many cases under the care of this physician, as well as from his own experience, Dr. Parkes considers that *calomel* is a medicine to be strongly recommended in typhoid fever. But it must not be given later than the tenth or eleventh day, and at no time in large doses.

One or two grains twice a day is enough, although Wunderlich gives one to five grains twice daily; but five grains is considered by Dr. Parkes to be too large a dose.

Dr. Wood, of Philadelphia, bears testimony also to the benefit to be derived from mercury about the seventh or ninth day of the fever. He believes "it tends in some degree to arrest the progress of the disease in the glands of Peyer, and to promote resolution of the inflamed patches. He prefers minute doses of the blue pill mass—a grain every two hours—till the mouth is slightly affected, associated with small doses of *ipecacuanha*, when the stomach is not irritable. The beneficial effect of this combination is shown by the tongue becoming moist, the skin relaxed, and the symptoms generally being ameliorated. Dr. Wood recommends twelve grains of *blue pill mass* to be combined with two grains of *ipecacuanha powder*, with two grains of *opium powder*; and the whole being divided into twelve pills, one may be taken every hour, or every hour and a half, or every two hours (*Practice of Medicine*, vol. i, p. 345, 4th edition).

Calomel is, however, contraindicated if the diarrhœa is excessive, or if there should be excessive pains in the bowels, with early and violent meteorism. It is also not proper to be given if the condition of the patient is anæmic, or if there is a decided hemorrhagic diathesis. It is most useful as a restorative of the intestinal functions in cases where the tongue is dry and coated, where thirst is absent, and when the urine is cloudy and of low specific gravity. If the first dose is vomited, the administration must be repeated. Calomel has no direct effect on the pulsation or respiration, nor on the cerebral functions; but its beneficial influence is very decidedly appreciable by the modifications of temperature which it induces, and which have been already noticed at page 360.

No general rules can be laid down to guide the treatment of the intercurrent phenomena or accidents of the disease. Combinations of remedies must be adapted to correct the several functions which may be simultaneously deranged, so that treatment must be varied according to the functions mainly implicated or suspended, and to the degree of their affection.

Abdominal pains and meteorism may be relieved by mustard poultices, or turpentine *stupes* may be applied, followed by simple hot water fomentations. After these remedies have been used, cold water compresses over the abdomen tend to lessen the tension and the gurgling in the intestines, and to diminish the tenderness on pressure. They counteract the inclination to meteorism, and lessen excessive diarrhœa. Dr. Huss believes also that the ulcerations in the ileum are prevented from spreading; and that perforations of the intestine have been of much rarer occurrence since he com-



emulsion with gum arabic, loaf sugar, water (Wood), or in an emulsion with the yolk of an egg and honey or mucilage (Huss). Amelioration of the symptoms may be observed in twenty-four or forty-eight hours—the tongue becoming more moist, and covered with a white fur—distension of the abdomen ceases to progress, and after a time diminishes. The use of the oil should be continued under these circumstances; but the dose should be gradually diminished.\*

[The chlorate of potash, in doses of five grains, may be given in camphor-water or weak bitter infusions, every two hours. Under its use the tongue often becomes clean and moist (GARNETT, HUNT, COPLAND, WATSON). Chlorinated soda—ten to fifteen drops in camphor julep—is highly praised by Copland, Chomel, and Graves.]

*Tonics and stimulants* may be absolutely essential on account of debility attending the advanced stage of the disease, generally about the third week. When the pulse is slow and feeble, the skin cool, the tongue and teeth incrustated with dark sordes, at an advanced period of the fever, then stimulants are obviously necessary. But even when the pulse is feeble, but yet frequent, and the skin hot, stimulants are even then known to be of service; but it is necessary to administer them with great caution, and to watch the effects constantly and closely. If their use is found to augment the heat of the skin, and to increase the frequency of the pulse, and to aggravate the delirium or stupor, it is then necessary to suspend their use. They are known to be doing good service, however, if they lessen the frequency of the pulse, and increase its fulness and strength; if the skin becomes cool and moist, and if the delirium is subdued or moderated; and especially if refreshing sleep be procured. Dr. Wood recommends the use of wine whey, prepared by adding *one quart of good sherry wine* to *two quarts of boiling milk*, and straining after coagulation. Of this a tablespoonful or more may be given every hour or every two hours. If the strength is greatly reduced, it may be necessary to give pure wine or brandy; or even sulphuric or chloric ether in cases of great prostration.

[If a patient with typhoid fever is properly nourished from the outset of the attack, alcoholic stimulants will not be necessary in a large number of cases. They are greatly overused. When, in spite of the early and regular administration of food, there is great prostration, or ataxic phenomena come on, stimulants should be at once prescribed; sound sherry wine is the best form for their administration, or, where diarrhoea is excessive, a pure Port, or Tarragona, wine; they are best given with milk, eggs, or broth. Iced champagne is often very grateful and happy in its effects, particularly where there is obstinate inappetency, or gastric irritability. Brandy is preferable to whiskey. It has been pro-

---

\* [The value of turpentine in typhoid fever is very doubtful. After a good deal of experience with the turpentine treatment, the writer has never seen any result which could be fairly attributable to it, except disordering the stomach. Dr. Gerhard's testimony is to same point; he says: "I cannot think it of much value . . . in ordinary cases it is perfectly nugatory." (*Pennsylvania Hospital Reports*, 1868.)—EDITOR.]





pure jelly of ripe fruits; but fruit in its crude state is to be strictly withheld. It is necessary, as a rule, to give food at certain intervals and in certain quantities. A wineglassful should be given at least every two or three hours [through the day and night,] according to the state of digestion and the demands upon the strength of the patient. It may be that the patient is unable to swallow, from the dry and shrivelled state of his tongue. Before offering him food or drink, therefore, the nurse should put a teaspoonful of lemon-juice and water into his mouth. She must then wait a minute or so, until the fur upon the tongue and mouth is softened and moist, after which the patient will often drink or take his food with ease. Milk in small quantities, [two or three ounces, to which a little lime-water, or bicarbonate of soda, or Vichy water, may be added, if there is much acidity of stomach, or cream, or a raw egg beaten up with water and sugar,] frequently repeated, will be found an excellent diet; and animal broths and jellies may ultimately be given. The *extractum carnis*, as prepared by Liebig, is a most valuable nutriment for typhoid fever patients.

[The patient should be placed under the most favorable hygienic conditions; he should be withdrawn from all disturbing or depressing influences, and perfect quiet of mind and body enjoined. Free and abundant ventilation and strict cleanliness of the apartment are indispensable, together with light bedding, which should be changed daily. The excreta must be removed immediately, and disinfectants and deodorizers frequently employed. Those parts of the body that may be exposed to pressure are to be daily examined, and if found reddened, they should be gently rubbed, and protected by some artificial cuticle, and a water- or air-cushion used. The state of the bladder should be ascertained frequently, and when necessary, the water ought to be drawn off.]

The utmost caution is necessary as to diet and aperients during convalescence; *first*, as to opening the bowels, castor oil or simple enemata are the only means which should be resorted to; *secondly*, as to diet, no flesh meat should be allowed till at least seven days after all the febrile phenomena have passed away, and the food should be as free as possible of excrementitious matter, [and at first should be given well hashed or minced.] Malt liquors should not be taken *before* food.

[During the whole of convalescence great care and vigilance are required, and the patient must be closely watched. Exposure to cold, or any fatigue or mental excitement, are to be strictly avoided. The quantity of food allowed must be rigorously within the capacity of the digestive function, otherwise there will be risk of gastro-intestinal troubles—inappetency, nausea, vomiting, gastric pain, tympany, diarrhœa, and intestinal perforation; or of evening fever exacerbations; or of a relapse. Bear constantly in mind the yet unhealed ulcers of the small intestine; strengthen the digestive organs; and be careful to do nothing that may weaken them. Most of the consecutive disorders of convalescence will gradually disappear as the waste of the system is healthily repaired. Paralysis, mania, dropsy, aphasia, if existing, cease by degrees as the strength returns. Wine, and the vegetable and mineral tonics are gen-



the tropical wars (Parkes "On the Causes of Sickness in English Wars," *Journal of Royal United Service Institution*, vol. vi). Wherever men are closely crowded together in ill-ventilated, unwholesome dwellings, typhus is sure to appear. It has often passed from the army to the civil population, and has thus dispeopled towns, and even great districts of country. But its ravages in the English army have never been comparable to those which have occurred in foreign forces, as the statements of Murchison and Parkes fully demonstrate: "In the year 1489 no fewer than 17,000 of the troops of Ferdinand, then besieging Granada, were destroyed by a spotted fever, to which the Spaniards applied the same name that they afterwards gave to typhus. In 1552 a petechial fever devastated the army of the Emperor Charles V during the siege of Metz. In 1556 the notorious '*Morbis Hungaricus*' appeared in Hungary in the army of Maximilian II, and thence spread over the whole of Europe" (MURCHISON, *l. c.*, p. 21). "In 1620 the Bavarian army in a few months lost in Bohemia not less than 20,000 men from spotted typhus; and the disease, being carried into other parts of Germany, obtained the name of 'the Bohemian disease.' In 1628 and 1632 the Swedish army under Gustavus Adolphus carried typhus into Northern Germany, and the population was so destroyed that, fifty or sixty years later, villages were left without inhabitants" (PARKES, *l. c.*). In the spring of 1643, while the Earl of Essex was besieging the town of Reading, this disease broke out in the army of the Parliamentary General, and in the garrison commanded by Charles I: it was communicated to the inhabitants of the surrounding country, and proved very fatal (MURCHISON, *l. c.*). The wars of Louis XIV were always followed by this disease, and the losses of the French army were enormous (PARKES). In 1799-1800 an epidemic of typhus occurred at Genoa, when the garrison was besieged by the French, and half famished; and the French army, during their retreat from Italy, communicated fever to the inhabitants of fifteen towns and villages where they halted on the route (FODERE). It was during the first fifteen years of the present century that the greatest ravages of typhus have been recorded, especially in the armies of Napoleon, and among the population of the countries which were the seat of war. It always became developed under circumstances of misery and privation, and was particularly prevalent and fatal among the inhabitants of besieged cities—as, for example, Saragossa and Torgau, Dantzic and Wilna, in 1803—and which told with such awful severity upon the famished French troops during the retreat from Moscow in 1812 and 1813 (MURCHISON). When Sir John Moore's army landed from Corunna, typhus became epidemic in the military hospitals in the south of England (Cheyne, *Dub. Hosp. Report*, vol. ii, p. 3). In May, 1812, the Bavarian army serving among the French numbered 28,000 men; in February, 1813, there were only 2250 men under arms. The great destroyer was typhus. In August, 1813, the first Prussian army consisted of 37,728 fighting men, having lost 16,000 men by the sword, and 10,000 men by disease, almost entirely typhus. In Mayence alone, of 60,000 French troops composing the garrison in 1813-14, there died of typhus in six months 25,000 men (MUR-



lished, says: "No case of typhus fever was reported during six months in 42,686 cases of all diseases amongst the Federal prisoners confined at Andersonville, notwithstanding that 40,000 men were crowded upon twenty-seven acres of land, and notwithstanding that all sanitary and hygienic laws were utterly neglected, and the earth was covered with abnormal human excrements and fragments of bread and saturated with urine, and the atmosphere was loaded with stinking effluvia. During the recent civil war I sought for typhus fever amongst the Confederate troops serving in the field, and amongst the general hospitals in various parts of the Confederate States; thousands of sick and wounded were examined with a view to the determination of the existence or non-existence of this disease amongst the Confederate armies; and even the prisoners confined upon Belle Isle, in the Libby Prison, and in Castle Thunder, in Richmond, Va., were not neglected in these examinations, and numerous medical officers of the Confederate army were interrogated upon this subject personally and by letter. No case of true typhus fever came under my observation during the war in any army, in any field hospital, general hospital, or military prison" (p. 600). He further states that the cases entered upon the Confederate sick reports as typhus fever were, in almost every case, if not in all, cases of typhoid fever occurring in those whose blood was scorbutic.]

**Phenomena and Symptoms.**—Typhus fever attacks persons of both sexes and of all ages, from early infancy to extreme old age, and its advent is somewhat sudden.

After a longer or shorter duration (generally a few days) of unpleasant sensations—in which general soreness, uneasiness, and fatigue without cause, loss of appetite, and disturbed sleep, are the prominent phenomena—the disease begins and advances gradually. It is not possible in all instances to fix the precise time of the commencement of the attack; but in the majority of cases the patient is seized with chilliness, which sometimes amounts to a rigor, usually followed by heat of skin, and occasionally by sweating, pains in the back and limbs, and frontal headache. This headache is a constant symptom, which ceases usually about the tenth day, and always before the fourteenth. During two or three days the chilliness and rigors occur at irregular intervals. The patient alternately hovers over the fire or desires to move from it; and although the skin at the time may be felt hot and burning, he still lingers near the fireplace, and yet again soon complains of the heat of the room; so that he feels when near the fire hot and oppressed, and when away from it chilly and uncomfortable. Loss of appetite, and more or less thirst, exist from the first; the tongue is white, large, and pale, but is afterwards covered with a yellow-brown fur, and is sometimes tremulous, indicating the early loss of muscular power and control. The bowels may be confined or regular; the urine is scanty and high-colored; and nausea with vomiting are often among the earliest symptoms. If sleep is obtained, it is disturbed by dreams, or by the occurrence every few minutes of sudden starts. It is consequently unrefreshing; and although the patient may have appeared to sleep for hours, yet he feels that he has not slept, and declares that he has never closed his eyes. This is the *coma-vigil* of Chomel. On the other hand, there is sometimes a constant ten-



and when the finger is firmly pressed on them, they grow paler, but do not entirely disappear. Thus they are said "to fade under pressure;" but they cannot be entirely obliterated, a stain of the cuticle remaining to indicate where they are. A still further change may take place in severe cases. The centres of the spots may become dark purple, unaltered in appearance by the firmest pressure, although their circumferences may fade; or the entire spot may change into a true petechia, becoming of a dusky crimson or purple color, quite unaffected by pressure, with a well-defined margin, and level with the surface. The spots of such an eruption are generally very numerous, close together, and occasionally almost covering the skin. Sometimes, however, they are very few in number, and situated at some distance from each other; and not to be distinguished at first from the *rose spot* eruption. The *mulberry eruption* usually occupies the trunk and extremities, but is occasionally limited to the trunk, and may now and then be observed to extend to the face. After the first, second, or third day after the eruption is apparent, no fresh spots appear, and each spot remains visible from its first eruption till the whole rash vanishes—that is, till the termination of the disease. When very numerous, the eruption, viewed as a whole, has not an equal depth of color. Some places are much paler than others, and the spots have a dull appearance, as if seen through the cuticle. A mottled aspect is thus sometimes given to the skin, on which the darker spots are seated; and hence (2.) A *subcuticular rash* has been also described, which is deepest colored on the most depending parts of the body. From this circumstance the eruption sometimes resembles *measles* so closely as to be distinguished with difficulty from the eruption in that disease. When the spots on the back are of a much deeper hue than those on the anterior surface of the trunk, the skin is at the same time so much congested at the back that slight pressure with the finger leaves a white mark, which slowly returns to its dusky red color. The eruption of the mulberry rash usually appears from the fifth to eighth day of this disease, and subsides between the fourteenth and twenty-first days (DR. JENNER).

Age seems to exert a considerable influence over the eruption; and the following rule has been laid down in relation to this modifying circumstance: In 100 typhus patients under fifteen years of age the rash will be absent in 25. In 100 typhus patients between fifteen and twenty-two years of age the rash will be absent in 14. In 100 typhus patients above twenty-two years of age the rash will be always present.

The spots of typhus fever continue ineffaceably persistent after death.

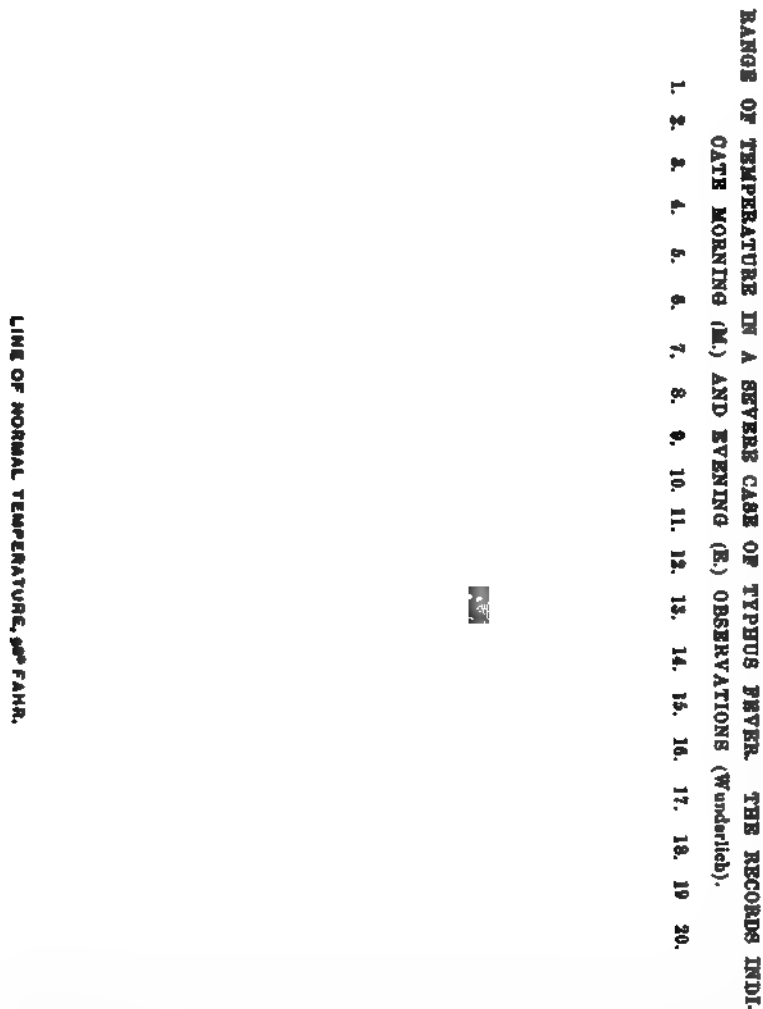
At the termination of the first, or commencement of the second week, the tongue has a large and swollen appearance, grows dry in the centre, and at the same time its white fur is replaced by pale dirty-brown mucus.

About the ninth or tenth day, or even earlier, the delirium becomes decided, sometimes violent, and always unquiet, although the attention may still be fixed by a sharp question. At this time the patient is in some cases violent, and, unless restrained, leaves his bed to





The skin throughout the whole course of typhus fever is often particularly sensitive, the slightest touch occasioning pain. The heat of the skin conveys also a burning pungent sensation—the temperature ranging from 102° to 107° Fahr. Greisinger, of Zurich, after insisting on the non-identity of typhus and typhoid fevers, has described the ranges of temperature in cases of typhus as particularly diagnostic and characteristic (*Arch. der Heilk.*, vol. ii, p. 557, 1861). Dr. Cheyne, of Dublin, recorded 109° Fahr. as the highest he observed, and a few days before death he observed the temperature to fall to 95° Fahr.



The course of typhus fever, although it may have some features in common with that of enteric or typhoid, yet shows great and numerous differences. In typhus, the fever, as denoted by the tem-



stage of the disease. The respiratory murmur at the same part becomes muffled, as if heard through a covering, and there is sometimes a little coarse, unequal crepitation. The urine, which is *now* secreted in large quantities—from three to four pints daily—is restrained, or passed into bed with the stools involuntarily. The skin is bathed in a profuse sweat, and the temperature is apt to fall below the natural standard. The patient lies on his back unable to move, or he sinks towards the bottom of the bed if his head be in the least elevated. Towards the middle or end of the second week a slough may form on the lower end of the spinal region, or on the region over the posterior spine of the ilium.

For a day or two before the fatal termination, the condition termed *coma-vigil* may come on. In this condition the patient never sleeps. He lies on his back with his eyelids widely separated, his eyes staring and fixed in vacuity, his mouth partially open, his face pale and expressionless. He is totally incapable of being roused to give a sign of consciousness, the breathing is often scarcely perceptible, the pulse rapid and feeble, or unable to be felt, the skin cool, perhaps bathed in perspiration. Life is only known to have ceased by the eye losing its little lustre, and the chest ceasing to effect its slow and feeble movements. Dr. Jenner has never seen recovery from this condition. Death generally takes place without any return to consciousness, and by syncope rather than coma (MURCHISON).

If the disease should terminate in recovery, the improvement in the condition of the patient is frequently sudden. Some time between the thirteenth and the seventeenth day he may fall into a profound quiet sleep, lasting for several hours; and generally after from twelve to twenty-four, or even more hours, he awakes decidedly improved in all respects—indeed, quite another man. At first he is bewildered or confused, and wonders where he is; but he may recognize his attendants and friends, and is conscious for the first time of his extreme debility. The complexion is clearer, the delirium has disappeared, the pulse has fallen in frequency and gained in strength, the conjunctivæ are no longer injected, the tongue is moist at the edges; there is perhaps a little appetite, the skin is softer and moist, and the spots paler. His limbs retain their sensibility, but when he attempts to move them, they seem at first as if separated from the body, so great is the prostration induced by typhus fever (MURCHISON). In a few days the tongue cleans completely, the appetite becomes ravenous and insatiable, and the patient rapidly regains strength. Dr. Jenner considers the duration of the disease to be measured by the duration of the eruption; and the average duration of cases that recover, he states to be from fourteen to twenty-one days; although, not unfrequently, in very mild cases, the fever terminates before the fourteenth day. After twenty-one days local lesions sufficient to cause death were always discovered in fatal cases of *typhus*. In other words, after the twenty-first day, death does not then occur *from the fever alone*, as may be the case before the twenty-first day. There are two very opposite circumstances under the influence of which the date of the first appearance of the



The occurrence of convulsions in such cases may be fairly referable, in the present state of our knowledge, to the morbid condition of the blood in *typhus* fever, and the altered condition of the nervous system which ensues; and probably they have always a uræmic origin. With reference to the absence of any appreciable lesion in the brain, as in these cases, it may be remarked that our usual instruments of research, applied to the nervous texture, are insufficient in all instances to indicate disease, even where it does undoubtedly exist. There are, for instance, physical conditions of texture, which are of the utmost importance in pathology, such as the specific gravity, and which are appreciable by the proper means and instruments of research, even when the tissue of the organ presents to our senses of sight and touch no external evidence of disease.

The cerebral complications are generally attended with what are commonly called "head symptoms," and are preceded by long-continued high temperatures. Dr. Jenner very emphatically calls attention to the fact, that the continuance of the headache complained of spontaneously after the commencement of delirium is generally indicative of increased vascular action within the cranium. It may also be noticed that the headache which precedes the delirium is often in such cases of a very severe and constant kind, the face being sometimes pale and sometimes red, and greatly expressive of the distress the patient suffers. The eye, haggard or brilliant, with its conjunctiva injected and its pupil contracted, is painfully sensible to the light, and is therefore generally closed. The least noise is insupportable, and the patient is troubled with noise in his ears. His temper is altered, and his answers short and fretful. This condition is that of increased excitement, but not as yet of delirium, and, supposing the membranes of the brain to be inflamed, denotes diffuse inflammation of those tissues. At the end of a period of time, varying from two to ten days, the patient becomes delirious. His delirium may assume every character,—joyous or melancholy, furious or tranquil; and in some cases he wanders from subject to subject, while in others he incessantly recurs to the same theme, and even to the same few words. In others, though the cases are few, the disease assumes every character of insanity; and, if permitted, the patient, confined in a strait waistcoat, presents the extraordinary spectacle of being able, in typhus fever, to walk about the wards. The phenomena of this stage show that the inflammation of the membranes of the brain has extended to the substance of the brain itself. The commencement of effusion is indicated by the active delirium changing into a low muttering (*typhomania*), by the patient no longer requiring restraint, by his muscles becoming spasmodically affected with slight twitchings, or *subsultus tendinum*, showing how rapidly the nervous power is exhausted, and how feebly supplied; also by the pupil of the eye becoming expanded or contracted; by the fæces being passed involuntarily; by the urine being retained; and by the rapid grouping of those other symptoms so happily described by Shakspeare as "the stony coldness of the feet creeping upward and upward," "the babble of green fields," and



and the nature of the complication is only to be recognized by careful physical examination and determination of temperature, which suddenly rises and continues high. The full expression of the morbid state is often for some time undecided; the exudation, being of a serous nature, is slow to solidify on the one hand, and yet the symptoms of resolution do not appear on the other. Dr. Hudson, of Dublin, attaches some importance to a certain tympanitic resonance, which becomes manifest over the diseased lung, as a sign of the existence of the pulmonic lesion. He describes it as "a tympanitic clearness over the solidified lung, without air being present in the pleura." Dr. Lyons explains this abnormal clearness as the result of the increased pressure of the respiratory column of air in the permeable portions of the pulmonary lobules, which become expanded beyond their natural volume, and thus a condition of temporary emphysema is produced, which yields a clear sound on percussion (Dr. Stokes, in *Medical Times and Gazette*, May 26, 1855).

In some cases of pulmonary lesions there appears to be a combination of circumstances which leads to a fluid or purulent state of the diseased part, resembling the third stage of pneumonia as described by Laennec. The conditions which lead to this form may be stated to be,—(1.) A sudden exudation and abundance of fluid matter; (2.) A great amount of tissue involved; (3.) Diminished vascularity and consequent (4.) Abeyance of absorption, tending to (5.) Increased fluidity of the diseased part; (6.) Breaking up or solution of the young and growing elements. A lung in this condition seems to have passed, as it were, at once into this state, without any well-marked hepatization.

**3. Gangrene of the Pulmonary Tissue** is by far the most formidable of the thoracic secondary lesions of *typhus fever*. The hepatization of the lung is not, as in the last instance, obscure, but the consolidation is at once sudden, complete, and extensive, involving perhaps the greater part of the lung, and coming on without any marked physical signs different from what are to be heard in the simple congestion of *typhus*. A gangrenous cavity forms in the substance of the solidified mass, and is only indicated by the fetid expectoration and the accompanying physical signs of a cavity. Large eschars are apt to form towards the pleural surface, surrounded with well-defined lines of demarcation where separation of the slough proceeds. In this gangrenous slough every simple element of the pulmonary tissue becomes disintegrated, almost perfectly liquescent; and sometimes it happens that the gangrenous cavity does not communicate with the bronchial tubes, and then the morbid state is difficult to diagnose, and its existence is often unknown till after death. With physical signs, the expression of the countenance of the patient is often highly suggestive. It suddenly becomes small, pinched, contracted, ghastly, miserable and death-like. The eyes are sunk and void of lustre; and, along with languor, the patient feels nausea, and sometimes vomits. There may be several distinct gangrenous centres, as if the lesion had been, from the first, disseminated or lobular.

**4. Secondary Cardiac Lesion.**—This lesion assumes the form which





gangrene of the lungs, convulsions, pyæmia, erysipelas, parotid swellings, inflammatory swellings, bed-sores, gangrene, renal disease, scurvy, the gouty diathesis.

**2. Combinations of Symptoms or Phenomena which may be regarded as of favorable import.**—(1.) A sudden fall in the frequency of the pulse; (2.) When a patient, after lying for days on his back, helpless and motionless, manages to turn himself round and sleep on his side, or if he is able to draw up his leg and rest it on the foot in the flexed position in the bed; (3.) Cases without rash, or in which the rash is scanty; (4.) When the excretion and elimination of urea and uric acid continue free and copious; (5.) Sudden cessation at the end of the second week of several of the unfavorable symptoms and phenomena; (6.) Diminution of the rapidity and increase in the strength of the pulse; (7.) A slight return of appetite, while the tongue becomes clean and moist at the edges; (8.) A diminution of the dusky tinge of the face, a less stupid appearance of the countenance, and a less injected state of the conjunctivæ, with signs of returning intelligence.

**3. Modes of Fatal Termination.**—(1.) Death during the primary fever may occur from syncope or from coma. In the former case the heart's action is enfeebled from paralysis or disease of its muscular tissue. In the mode of death by coma the blood has undergone such modifications as render it incapable of supporting the changes essential to existence. Its contamination seems mainly due to the admixture of urea and other products of the retrograde metamorphoses of tissue, and from the diminution and destruction or solution of its red corpuscles. (2.) Death is for the most part due to a combination of syncope and coma; and, as a rule, the patient is quite unconscious for a considerable time prior to death. (3.) Death may occur from one of the many complications which happen before or after the cessation of the primary fever.

**Morbid Anatomy.**—The morbid anatomy of cases of typhus fever has been carefully investigated by Gerhard and Pennock, A. P. Steward, John Reid, Thomas Peacock, William Jenner, Felix Jacquot, Barrallier, and Murchison. All are agreed that there is no constant nor characteristic lesion; and they may be summed up generally as follows: "A fluid condition of the blood; hyperæmia of the cerebral membranes and increase of intra-cranial fluid; bronchial catarrh and pulmonary hypostasis; softening of the heart, liver, spleen, and pancreas; hyperæmia and hypertrophy of the kidneys" (MURCHISON, p. 245).

**Treatment of Typhus Fever.**—Before considering the treatment of typhus fever, it is of the greatest importance to be aware of the changes which go on in the system during its progress. Dr. Parkes has observed the nature of these changes in a most conclusive manner. His observations are of great scientific interest, and of important practical bearing ("Gulstonian Lectures," in *Medical Times and Gazette* for February 28, 1857). In an uncomplicated case of typhus fever the body loses flesh rapidly, owing not only to diminished ingress of food, but also to increased egress of bodily structures in the form of excretory products. The metamorphosis of tissue,



*typhus* and *typhoid* fevers are not exceptions) may be summed up as being a combination of measures to *reduce excessive heat*, to insure *proper excretion*, and to *act on the semi-paralyzed nerves*; and, as Dr. Murchison justly observes, “every remedial agent which shall be found to promote the elimination of urea, without increasing the destructive metamorphosis of tissue, will deserve a trial in typhus” (*l. c.*, p. 268).

To reduce heat and to regulate elimination are but secondary indications in the treatment of *typhus* fever, compared with the influence which must be exercised over the nervous system; and one of the greatest objects of therapeutics at the present day is to find substances which will act on the nerves and the blood, and restore them in some way to their normal action. (See page 153, *ante*.)

**Special Indications for Treatment.**—Our objects in the treatment of typhus fever should be,—(1.) To neutralize the poison and to correct the morbid state of the blood; (2.) To eliminate the poison and the products of the destructive metamorphosis of tissue; (3.) To reduce the temperature; (4.) To sustain the vital powers, and to obviate the tendency to death; (5.) To relieve the distressing symptoms; and (6.) To avert and subdue local complications (MURCHISON, p. 265).

1. In the belief that the morbid condition of the blood in typhus fever may be due to the presence of ammonia in some as yet unknown combination, the use of mineral acids has been recommended by many physicians. Murchison considers their beneficial effects in typhus as undoubted, and in this opinion he is confirmed by the experience of Huss of Stockholm, Haller of Vienna, and of Mackenzie, Chambers, and Richardson, in this country. Huss recommended phosphoric acid in doses of ten to fifteen drops every second hour, believing that the phosphorus exerts a special influence on the brain; but in the advanced stage, and especially if sweating, numerous petechiæ, or ecchymoses be present, he has recourse to sulphuric acid in doses of fifteen to twenty drops every hour or every second hour. Hydrochloric acid is preferred by Drs. Murchison, Richardson, Mackenzie, and Chambers. It may be given to the extent of one fluid ounce of the dilute acid, mixed in a quart of barley-water, sweetened with syrup of ginger, and flavored with lemon-peel. Dr. A. P. Stewart has used with advantage the *tinctura perchloridi ferri*, in doses of thirty minims every three hours. Dr. Murchison recommends *nitromuriatic acid*. He prescribes twenty minims of *hydrochloric acid* with ten minims of *nitric acid* every three hours, each dose being diluted with the patient's drink. But if the “typhoid state” is developed in a marked manner, *dilute sulphuric acid*, in doses of fifteen to twenty minims every three hours, in combination with *ether*, and small doses of *quinine*, are to be had recourse to as in either of the following formulæ:

R. Acid. Hydrochlor. dil., ℥xx; Acid. Nit. dil., ℥x; Spt. Æther. Nit., ℥lx; Liquor. Cinchonæ, ℥xxx; Decoc. Scopar. comp., 3j; *misce*. A draught so composed may be administered every third hour.

Or, R. Quiniæ Sulph., gr. ½; Acid. Sulph. dil., ℥xx ad ℥xxx; Æther. Sulph., ℥xv ad ℥xxx; Syrup. Aurant., ℥lx; Decoc. Scopar. comp.,



Dr. Murchison recommends that large quantities of this salt should be given with the beef tea.

The action of the bowels is to be maintained by emetics and laxatives. In the first instance, if the patient is seen early—*i. e.*, before the sixth day—an emetic of ipecacuanha (one scruple), and of antimony (one grain), or of carbonate of ammonia (two scruples), in place of the antimony, is to be administered. If the bowels remain confined after the emetic, a mild laxative of rhubarb and calomel, or of castor oil, is to be given; and failing these, or in place of them, a simple enema is to be administered (MURCHISON, p. 269). The advantages of emetics are, that they relieve the patient to some extent by mitigating or removing headache and general pains. They also reduce the temperature, abate thirst, and quiet gastric disturbance.

Emetics, however, are contraindicated if the patients are unusually weak, or if the disease has advanced beyond the first week. Laxatives and enemata, however, ought to be repeated daily, if required, so as to secure a motion of the bowels once a day. In this respect the treatment is different from the treatment which ought to obtain in typhoid fever, as already mentioned. Excrementitious matters in the intestines must be removed by gentle aperients. The dark offensive matters accumulated in the intestinal canal in typhus fever may have a secondary deleterious effect on the system if they are allowed to remain. Purging, however, is to be avoided, and *fresh-made* compound rhubarb pill mass, which *tends to stimulate the peristaltic action of the intestines*, is as good a medicine as can be given, followed, if necessary, or alternated, by a small dose of castor oil, or by a simple enema.

Diaphoresis is not to be encouraged beyond the insensible transpiration of the skin; and to remove which the wholesome detergent of tepid water sponging is most beneficial. It ought to be used twice or three times daily, and quantities of *Condy's fluid* or of *muriatic acid* (3j ad Oj) may be mixed with the tepid water (MURCHISON). The measure is a good one in a hygienic point of view, and it contributes—

3. To reduce temperature, for which the external application of cold water was once practised to an extreme degree by Currie, as originally recommended by Dr. Robert Jackson. In health such an application as that of cold water has a great effect in reducing temperature, and tends to increase metamorphosis (LEHMANN, SANDERSON).

4. The vital powers are to be sustained by food in the first instance. For this purpose, nourishment ought to be given often, and at stated intervals—at least once every three or four hours after the fourth day of the fever. Even if the patient is asleep, or seems to be so, he must be roused at these stated intervals (not oftener) to take his food or his stimulants. But if, towards the period of the crisis, the patient appears to be in a sound sleep, he ought not to be disturbed. The indications for treatment just described apply to the earlier stages of the fever, up till about the fourteenth day.



“typhoid state”—are indications for the liberal administration of alcohol; but the propriety of giving stimulants in delirium depends on the state of the pulse. If, on the trial of stimulants, the patient becomes tranquil, they do good, and may be continued; if the reverse, their use must be suspended.

(g.) A dry brown tongue is an indication for wine or brandy, and if it becomes clean and moist at the edges under the use of either, such stimulation is beneficial.

(h.) Complications, as a rule, increase the necessity for stimulation; and large quantities of stimulants are called for if *pyæmia*, *erysipelas*, *bronchitis*, *pulmonary hypostasis*, *pneumonia*, *inflammatory swellings*, *bed-sores*, or *local gangrene* should supervene.

(i.) Persons who have led intemperate lives, and old persons, require stimulants early in the fever, and in large quantities.

The effects of alcoholic stimulation require to be most carefully watched throughout the whole period of their administration. Four ounces of wine in the twenty-four hours is enough to begin with; for if the blood be overloaded with the products of alcoholic ingestion, further alcoholic stimulation will lead to increased contamination, and it is rare that more than eight ounces of brandy in twenty-four hours are necessary.

There are differences in the demand for stimuli in the typhus of different countries, and in the fever of different epidemics. Dr. Wood tells us that in America cases requiring wine or brandy are extremely rare. Dr. Stokes says that the *typhus* in Ireland demands large quantities of wine. In Scotland, also, wine is the great mainstay in the treatment of *typhus fever*, requiring often to be administered largely.

Port, Sherry, Marsala, Madeira, brandy, gin, or whiskey, possess no peculiar advantages apart from the alcohol contained in each. Spirits contain from fifty to sixty per cent. of alcohol, Sherry and Port from seventeen to twenty-four per cent., and malt liquors from six to eight per cent. Two fluid ounces of spirit will thus be equal to five or six of wine, and spirits ought to be given diluted; and if the prostration is great, and when the skin is cold, and covered with perspiration, the best stimulant is brandy or whiskey punch, given as hot as it can be taken, in small quantities at a time, frequently repeated. In urgent cases stimulants ought to be given every hour; and, as a rule, a larger quantity will be required during the night and early morning than in the daytime, for it is usually towards morning that temperature tends to get low, and the vital powers are at their lowest ebb (MURCHISON). At the same time it must ever be remembered, as Dr. Jenner justly observes, that “in no disease is the advantage of refraining from meddling more clearly displayed than in typhus fever; and in no disease is the prompt use of powerful remedies more clearly indicated. It is in determining when to act, and when to do nothing, that the skill of the physician as a curer of disease, in the case of fever, is shown. Interfere by depletion or by stimulation when nothing should be done, and the patient is lost, who, if it had not been for you, would have been safe. Refrain from depletion or withhold stimulants





subjects two or four leeches may be applied to the temples; and in aged or infirm persons warm fomentations to the head are advisable (GRAVES and MURCHISON). But if anæmia is the cause of the headache, as may be suspected from the state of the vascular system, then stimuli are called for. Four to six ounces of wine may be given in divided doses during the day and night of twenty-four hours. If the pulse continues to get weaker, the wine must be increased.

The headache of typhus naturally abates about the eighth day; but it is sometimes rendered worse by sleeplessness; and if the remedies for the headache do not relieve it, nor tend to induce sleep, then opiates may be given, combined with antimony, if the skin be dry and hot and the pulse of good strength. Dr. Murchison thinks that the employment of opium in typhus is more dreaded than it ought to be. The dose of opium should be given about 9 P.M., followed in two hours by half the dose if the patient does not sleep. The form of the opiate and dose may be ten to twenty minims of Battley's solution of opium, or fifteen minims of the solution of the *bimeconate of morphia*, or five grains of the *opium pill* of the *British Pharmacopœia*. Dr. Murchison teaches us to distinguish two forms of delirium as a guide to the administration of opium, combined with antimony in the one form, and with ethereal stimulants in the other. When the condition of the patient approaches more to that of *delirium ferox*, the cardiac and radial pulses being of good strength, after trying the cold affusion, and remedies already mentioned, then opium combined with antimony ought to be given without delay, as in the following prescription:

R. Liq. Opii. Sedat. (Battley's), ℥℥x; Antim. Tart., gr. j ad gr. ij; Aquæ Camph. ℥vj; *misce*. A large spoonful of this mixture is to be given every hour until sleep is induced.

On the other hand, if the delirium approaches in its character that of *delirium tremens*, the radial pulse is usually quick and feeble, the cardiac impulse diminished, and the first sound of the heart more or less inaudible, then the opium must be combined with alcoholic or other stimulants, the amount being regulated by the state of the pulse and heart. Dr. Murchison suggests the following prescription:

R. Liq. Op. Sed. (Battley's), ℥ss.; Spt. Ætheris, ℥℥x; Aquæ Camph., ad ℥ij; *misce*. Commence by giving two table-spoonfuls of this mixture, and repeat it every hour till sleep is obtained.

Or opium to the amount of half a grain may be combined with three grains of camphor in a pill, and such a pill may be repeated, if necessary, every two hours.

Cases requiring such treatment ought to be seen at least three or four times daily. If dyspnœa is urgent, and lividity of the face betoken pulmonary lesion, defective arterialization of the blood, and venous congestion of the brain, opium in any form must be withheld; and it must likewise be discontinued if any tendency to stupor



recruiting. Commissions or commands of regiments were wont to be given to those who collected a certain number of men. Every low purlieu, every infamous haunt, every jail even, used to be ransacked for recruits. Wherever these men went they carried typhus, at that time the constant scourge of our towns and our jails; and complaints of the introduction of typhus fever from this source are frequently found in the writings of army surgeons of the last century. In connection with this point, Dr. Donald Munro, in 1764, gives the following caution: "That particular regard be paid to those soldiers picked up in the streets, or who have been taken out of the Savoy or other jails. All dirty rags from such people should be thrown away or burnt" (Dr. PARKES, *l. c.*). There is now ample proof that typhus fever may be communicated *by fomites* adhering to apartments, articles of clothing, and the like; and, provided fresh air be excluded, it is known that such articles will retain the poison for a very long time. Herein lies a fallacy which pervades the argument from cases to prove the generation of the disease *de novo*. The poison may be said (like that of small-pox) to be constantly in existence. Dr. Murchison quotes some striking instances of the propagation of typhus fever by *fomites*. For example, he refers to the instance related by Foderé, in which the soldiers of the French army, during their retreat from Italy in 1799, communicated fever to the inhabitants of towns and villages where they halted on their route, although the army was not attacked by fever, and soldiers travelling *singly* did not communicate the disease. But as he omits to connect this with the fact that typhus prevailed to a great extent in the towns they besieged, and in some instances obtained possession of, the source of the fomites is not made apparent, and therefore in my last edition I was made to misrepresent this instance given by Dr. Murchison, and to put it forth as an example of generation *de novo* (see p. 87 of his work *On Continued Fevers*). He quotes, however, the recent instance of the Egyptian vessel, the "Scheah Gehald," at Liverpool, the crew of which disseminated the poison of typhus by their clothes and persons, although, as he says, they had not the disease themselves. But this is an error. The careful investigation made by Dr. Parkes into the history of this epidemic on board the Egyptian ship clearly shows that the crew suffered from typhus fever (*Statistical, Sanitary, and Medical Reports of the Army Medical Department* for 1860, p. 359). The facts of the case have been curiously confused; but the following statement, from the above and other sources, may be relied on: A number of men (476, chiefly Arabs) were shipped on board the "Scheah Gehald" at Alexandria, to proceed to Liverpool to navigate back a man-of-war then in that port. The weather was cold and stormy, the hatches were battened down during a lengthened voyage of thirty-two days from Malta; and the men, unaccustomed to the rigor of a Northern winter, and not provided with suitable clothing, crowded below for warmth and shelter. Even they whose turn it was for duty had to be driven up on deck. They were extremely crowded on board, and the space below deck was quite insufficient for so large a number (for the crews of two vessels were on board); and there was no at-



haps in their clothes—and communicated it to the attendants. The remaining crew (350) of the ‘Scheah Gehald’ were sent to Alexandria on board the ‘Voyageur de la Mer.’ The people of Liverpool were probably so glad to get rid of them that they did not take the trouble to see that the typhus fever had been eradicated, and several of the men were sent at once from the Southern Hospital. The ‘Voyageur de la Mer’ lost some men on the passage, and landed several at Falmouth, and some with unequivocal typhus at Malta; and of thirteen Englishmen who were in her, six took the disease.”

“The case of this Egyptian vessel,” continues Dr. Parkes, “afforded almost the best opportunity seen in this generation for the investigation of the important question of the spontaneous generation of typhus. The opportunity was, however, lost. That all the circumstances which have been supposed to be capable of calling into existence the specific poison of typhus were present in this foul and filthy ship is clear; but every one who reads all the published statements will at once perceive that one link of the chain of evidence is wanting, and that it has *not* been proved that some of the crew were not ill with typhus when they embarked at Alexandria, or became ill within the incubative period. On the contrary, the interpreter informed Mr. Pemberton that some of the men were sick when they came on board. It can never now be ascertained whether there were such cases or not, and the history of the outbreak at Liverpool affords another instance of the loss of a great opportunity for definitely setting at rest a most important question.” The case of the “Scheah Gehald” now assumes exactly the same aspect as many instances historically quoted as examples of generation *de novo*—namely, that however plausible may seem the probability, there is *no proof* that typhus fever arose *de novo*. Seeing that such is the state of the question as to the origin of typhus—that it is exactly in the same state as our knowledge regarding the origin of small-pox or of typhoid fever—that it has been in existence from the earliest periods of the world’s history—that it is undoubtedly propagated from pre-existing *foci*, and by continuous succession, the immediate direction of investigation ought to bear especially on the following points, namely: How long can the typhus poison exist or be maintained in a condition fit to assume activity under favorable circumstances? What is the distance at which it is potent? Has temperature any influence upon it? What are the conditions or combination of circumstances more or less essential to the development and propagation of the typhus poison.

The fact that typhus fever is contagious is based on evidence which shows,—(1.) That, when typhus commences in a house or district, it often spreads with great rapidity; (2.) That the prevalence of typhus in single houses, or in circumscribed districts, is in direct proportion to the degree of intercourse between the healthy and the sick; (3.) That persons in comfortable circumstances, and living in localities where the disease is unknown, are attacked on visiting infected persons at a distance; (4.) That typhus is often imported by infected persons into localities previously free from it; (lastly), That its contagious nature is indicated from the success



poison may also adhere to the walls of dwellings, to beams of wood, and to articles of furniture. Dr. Murchison quotes an account by Pringle of twenty-three persons being employed in refitting old tents in which typhus patients had lain; and seventeen of these persons died of the infection. He also refers to an observation of Lind, who mentions several instances in which infected ships continued to impart the disease long after the sick had been removed. Similar cases are recorded by Jacquot respecting the Crimean typhus.

Nurses and other attendants in fever hospitals are well aware of the danger of contracting typhus from infected clothes, and from cleaning the bedding of the sick; and in some instances they are in the habit of "measuring the amount of danger by the badness of the smell." Thus they are liable to contract typhus fever without having had any direct communication with the sick. With regard to the kind of clothing most apt to retain and convey the specific poison, woollen textures are found to be the most dangerous. Haller, of Vienna, has made experiments on this point. He observes that *dark-colored* materials of clothing are more apt to absorb the contagion of typhus, and to convey it to other individuals, than those which are light-colored. He found that, among troops wearing dark-colored uniforms, it more frequently happened that new cases of typhus entered the hospital after a convalescent patient joined his corps, than those wearing light or white uniforms. The fact has been often observed, that in dissecting-rooms dark clothes acquired the cadaveric odor sooner, and were deprived of it less readily than light ones; and he ascertained by experiments that the absorption of odors is regulated by the laws which govern the absorption of light. Haller also found that the specific poison of typhus fever is lighter than atmospheric air. When the under stories of an hospital were filled with typhus patients, those in the upper stories were always observed to become infected when there was a communication between the air of the two stories. On the other hand, when only the upper stories contained cases of typhus, the patients in the under part of the house enjoyed perfect immunity (*Edin. Med. and Surg. Journal*, 1853). Dr. Murchison has observed that, if the poison be very concentrated, the length of the period of exposure sufficient to contract the disease is very brief—not more than a few minutes; and the latent period during which it remains in the body, without betraying its presence in any way, has been very variously estimated. Nine days is the result of Dr. Murchison's observations. Instances, however, are not uncommon in which the disease manifests itself almost instantaneously after exposure to the poison. In such cases these extremely susceptible persons are generally conscious of the peculiar and offensive pungent odor emanating from the beds or bodies of the sick. They are generally then immediately seized with prostration, nausea, rigors, and headache, followed by the regular development of the disease. Such persons are thus almost conscious of the moment at which the poison entered their system. On the other hand, the length of time between exposure and attack may be greatly prolonged. In my own case, I was three months in daily and close attendance in the fever





Sometimes it has been described as a variety of a well-known form of fever, and at other times as a new disease.

In Scotland in 1817-18 this fever was clinically recognized and described by Drs. Christison and Welsh; and when it reappeared as an epidemic in Edinburgh and Leith in 1843, Dr. Christison had no difficulty in again recognizing it. About this time it also appeared in Glasgow as an epidemic about a month before its outbreak in Edinburgh; and subsequently it became prevalent in Dundee and other large towns in Scotland. It was observed with great accuracy, and its phenomena were recorded in the medical journals of the period, by Drs. Craigie, Alison, Arrott, Henderson, Douglas, Jackson, Mackenzie, Cormack, and Wardell. It formed a part of the fever epidemic of Ireland in 1817-18-19, described by Barker and Cheyne; and it had been prevalent in Ireland for many years. Epidemics of it were described by Rutty, in his *Chronological History of the Diseases of Dublin*, as early as 1739 and 1741. In most of the periods of epidemic fever referred to, the commencement of the epidemic was characterized by the greater preponderance of cases of relapsing fever; and as the epidemic advanced, the number of cases of relapsing fever gave place to a preponderance of typhus cases (STEELE, R. PATERSON, ORMEROD, MURCHISON). In 1847 it became again epidemic in Glasgow, Edinburgh, and the large manufacturing towns of Scotland, as well as in London, when it was carefully described by Dr. Jenner, who, moreover, shows that its characters have remained constant since they were first described by British physicians. During the same year it prevailed in some parts of the Continent, and more especially in the Prussian province of Upper Silesia, and in some other parts of Germany. There it has been described by Virchow, Bärensprung, Dümmler, and Suchanek. These observers, however, did not know or recognize the fever so well and precisely described by the Scotch physicians; and, indeed, Dr. Parkes, was the first to indicate, in his admirable paper on "The Diagnosis of Fevers," already noticed, that the epidemics these German physicians described were mainly made up of the relapsing fever. This fever evidently formed the great bulk of the cases. Yet, although its characters are thus so striking that the most superficial observer could not fail to recognize them, the German systematic writers (except Virchow) make no allusion to relapsing fever as a separate and distinct disease; and even those who observed the fever in Germany failed to draw that obvious inference to which the Scotch physicians unanimously came—namely, that *relapsing fever* is a disease altogether distinct from *typhus* and from *typhoid fever*. If it is not so, "we know not," as Dr. Parkes observes, "that any medical evidence whatever can be relied upon."

In the summer of 1855 it prevailed, after the hardships and privations of the preceding winter, among the British troops in the Crimea, where it was recognized and described by Dr. Lyons. It has not been observed in France, nor in any other part of the continent of Europe.

The observations of Dubois, Austin Flint, and others, leave no



which he saw in South America. It broke out in Peru in 1854, and proceeded along the chain of the Andes, never at a lower elevation than 1500 metres, until in 1859 it had reached Bolivia and the neighboring parts of Chili. In about 1000 cases the deaths were 250, equally divided among both sexes—children under 16, 92; adults, 110; over 60, 48 (*Gazette Médicale*, 1865).]

In India and in all tropical countries it is as yet unknown.

Since the epidemic of 1847 and 1848, Dr. Murchison writes that relapsing fever has been gradually disappearing; and for the seven or eight years previous to 1863 not one case has been observed in the hospitals of Edinburgh, Glasgow, or London. Professor W. T. Gairdner has not seen or heard of a single case at Edinburgh since 1855; and, according to Drs. Lyons and McEwen, true relapsing fever has of late years been a rare disease in Ireland.

Like other continued fevers, its specific cause is unknown; but it selects its victims from the poor and ill-fed, who live miserably, in crowded, filthy, ill-ventilated apartments, rather than from the wealthy and well-fed, who live in comfort and in well-aired abodes. Its poison appears to be of a specific kind, and the phenomena of the fever are very different from those of *typhus* and *typhoid fever*. Patients recovering from either *typhus fever* or *typhoid fever* may catch, by contagion, the *relapsing fever*, while patients convalescent from *relapsing fever* may also take either of the forms of *continued fever* already described. It has been supposed by some (Dr. CORMACK) to be identical with the malarious form of *yellow fever*; but there is not sufficient evidence to establish the point. It seems more nearly to approach in its nature some forms of remittent fever, on account of the repetition of the rigors, often at regular daily periods, for two or three days. The marked periodicity of its relapses, which “come on like a fit of ague almost to an hour” (Dr. R. PATERSON), and the enlargement of the spleen to a greater extent than in any other form of fever (JENNER), point also to a malarious origin. On the other hand, epidemics of relapsing fever, as Murchison shows, appear to commence, progress, and decline quite irrespectively of the season of the year.

The evidence that a specific poison exists and is formed in cases of relapsing fever, and when so formed is communicable from the sick to the healthy, rests on evidence similar to that adduced in cases of typhus; and the same objections may be taken to the evidence which aims at establishing the spontaneous generation of the specific poison. There are causes, circumstances, or conditions which obviously favor the accession of relapsing fever, and no doubt also, its occurrence in an epidemic form; and chief amongst these predisposing causes must be placed destitution and want of food, while the names applied to the disease by different countries indicate the popular belief as to such predisposing causes being credited with originating the disease in the first instance. Thus it is spoken of as the *famine fever* of the British Isles, and the *hunger pest* of Germany.

**The Primary Paroxysm.**—The seizure is generally, indeed almost



large proportion of cases there is decided jaundice, and in others the skin exhibits a bronzed hue. The jaundice is not attributable to any obstruction of the *ductus communis choledochus*, as bile passes freely, and even copiously, with the stools, and as, after death, the gall-duct is pervious. There is generally tenderness over the region of the liver in such cases; and it may be enlarged. Thirst is excessive; the appetite absent or voracious, and the bowels constipated. The tongue, at first moist, is covered with a white or yellow fur, which it may retain throughout the illness; and, in many cases, it may become dry all over, or with a brown dry streak down the centre, after the third or fourth day.

**The Crisis.**—After the patient has continued in this state for a period varying from five to eight days, a sudden change takes place, immediately preceded, in most cases, by an exacerbation of all the symptoms. “When every symptom appears hourly becoming graver—when the restlessness and general distress have reached their highest point—then ensues a most remarkable series of phenomena, followed by a remarkable intermission of all the symptoms, and an apparent restoration to health.” This period has received the name of “Crisis,” and supervenes generally on or about the *seventh* day, and its advent is rarely prolonged beyond the *eighth*. This change is ushered in by a most profuse perspiration, in some instances with an eruption of miliary vesicles, which breaks out from the whole surface of the skin, and in the course of a few hours the patient appears nearly well. More rarely the change is indicated by epistaxis as well as by perspiration, or by profuse diarrhoea, catamenial discharge, or hemorrhage from the bowels; and after either or all of these apparently critical changes have been established for a few hours, there is a complete and abrupt cessation of all the bad symptoms. The pulse quickly regains the natural standard, the tongue cleans, the appetite and sleep return, and the countenance resumes its tranquillity. This alteration is very often effected within a few hours, and on the following day the patient generally considers himself in all respects quite well, and may so continue to improve rapidly for *four* or *five* days. During this period, however, there are some patients who suffer from violent muscular pains in the limbs.

**The Relapse or Recurrent Paroxysm.**—About *seven* days after this critical change, or between about the *twelfth* to the *twentieth* day from the commencement of the illness, but generally on the *fourteenth* day, a sudden relapse occurs “in ninety-nine cases out of every hundred.” This relapse commences suddenly, like the first seizure, by rigors, headache, loss of appetite, vomiting of green fluid, which is quickly followed by a hot skin, quick pulse, and a coated white tongue, confined bowels, followed by delirium, so that the phenomena may be exactly represented as a repetition of the first attack. In the interval of convalescence between the first and second attacks the pulse often becomes slow to an extreme degree, as slow even as forty-five to sixty beats in the minute; but, suddenly, on the relapse commencing, it again rises to 120 or more. In ordinary favorable cases perspiration would again occur in *two, three, four, or*



paroxysm and one relapse—the total duration of the fever extends to about *three weeks*; and the convalescence is very slow—much slower than in typhus. The *relapsing fever* is very exhausting in its effects upon the constitution; and, dating the period of convalescence from the termination of the last attack, the time taken to recover is in most cases unusually long. To those, indeed, who suffer from more than one relapse, it is almost impossible to have health completely restored for a long time. They become a prey to various sequelæ of fever, or they continue sickly for months, with pallid countenances, puffed ankles, palpitations, extreme debility, noises in the ears, dimness of vision, diarrhœa, or dysentery. Dysuria is a frequent complication amongst women during the relapse. In many instances during the epidemic of 1847 and 1848 in Ireland, convulsions occurred in cases which otherwise seemed to be progressing favorably, and death invariably followed them. Dr. William Robertson observed in Edinburgh (and the Irish physicians record a similar observation) that delirium of a violent character occurred during convalescence, or after the critical discharge had taken place. It generally came on suddenly, with incessant talking, a rapid weak pulse, followed by perfect unconsciousness, flushed face, and contracted pupil.

[**Anatomical Characters**].—No special anatomical lesion has been pointed out as peculiar to *relapsing fever*. The most constant lesion is enlargement of the spleen, the size attained by that organ being on the whole larger than in either typhus or typhoid fevers. Dr. Jenner has recorded the weight in one case to have been as much as thirty-eight ounces, and of a size in proportion. Its substance is generally softened, sometimes diffuent. It is usually seen at its largest size when death occurs during the final paroxysm; but if death occurs during convalescence, the spleen is of a normal size. Occasionally pale, red, fibrinous infarctions are found in its substance and near its surface. They are easily broken down, have a fine granular fracture, and are considerably firmer than the surrounding tissue, from which they are separated by a distinct line of demarcation. As a rule, there is but little congestion of the lungs, the weights of which contrast singularly with the weights of organs in subjects dead of typhus fever.

The blood in a few cases has been found fluid throughout the body after death; but generally, when drawn from the body during the febrile paroxysm, it is buffed; and decolorized coagula are found in the heart and large vessels after death more frequently than in cases of typhus. In several cases urea has been detected in the blood in considerable quantity. The proportion of white corpuscles is increased, a fact of interest in connection with enlargement of the spleen, and the state of anæmia so commonly observed (CORMACK, ALLEN THOMSON, MURCHISON). The liver is generally large, and the gall-bladder filled with dark thick bile.

**Sequelæ of Relapsing Fever.**—One of the most common results is the occurrence of excessive pains in the limbs, more especially expressed about the knee and ankle joints; and even the long bones appear to be the seat of these pains in some cases. Combined with





with a little syrup. This quantity is to be used up during the twenty-four hours. Acetate of potash and nitric ether may be used for the same purpose; but the nitre has the additional advantage of keeping open the bowels.

The surface of the body should be frequently sponged over with cold or tepid water; stimulants are not usually necessary, but they may be required in the stage of languor or exhaustion ensuing on the crisis; or in cases where great debility has preceded the attack. If any anæmia exists, or if an anæmic murmur can be detected, stimulants must be given early. When jaundice appears, Dr. Murchison recommends that nitro-hydrochloric acid should be given in combination with nitre, as in the following formula:

Twenty minims of hydrochloric acid, with ten minims of nitric acid, every three hours, each dose diluted with the drink of nitre and barley-water already prescribed.

Contamination of the blood with urinary products is the great danger in cases of relapsing fever; and therefore, in all cases of relapsing fever, particular attention must be paid to the state of the urine, especially towards the period of the first crisis. When the daily amount is much reduced, or if entire suppression should ensue, and particularly if stupor, confusion of thought, or drowsiness should supervene, the bowels are to be freely moved by compound jalap powder, or by a turpentine enema. Determination to the skin should be promoted by the hot air bath; and saline diuretics may be given every two or three hours (MURCHISON). No means hitherto discovered will prevent the occurrence of the relapse.

### FEBRICULA.

LATIN, *Febricula*; FRENCH, *Fièvre éphémère*; GERMAN, *Febricula*; ITALIAN, *Febbricola*.

**Definition.**—*A simple fever in which the expression of the febrile phenomena is of very short duration, lasting, as a rule, for twenty-four, thirty-six, forty-eight, or seventy-two hours or more, attended with a frequent, full, and often firm pulse, white and coated tongue, pains in the loins and limbs, thirst, constipation, a scanty discharge of high-colored urine, hot and dry skin, sometimes an eruption of roseola or erythema about the loins or thighs, coming and disappearing with the fever (MOREHEAD); severe headache, sometimes acute delirium, and flushed face. The subsidence of the fever is generally associated with copious perspirations, or herpetic eruptions.*

**Pathology.**—We do not know of any specific poison as the cause of such phenomena as those detailed in the definition; neither have we any evidence that febricula is a contagious or miasmatic disease. There are many different causes which are known to be capable of exciting expressions of febrile phenomena similar to those mentioned in the definition,—such as exposure to great heat or cold, surfeit, inebriety, mental or bodily fatigue or excitement; and specific poi-



own observations, the amount of urine is extremely small (twelve to twenty ounces), of very high specific gravity (1035–1037), with the solids and sulphuric acid very much over the average, and the amount of urea large. When the temperature falls, the quantity of urine rapidly augments. The increase of urea and of the solids is not so much, however, as in the height of the more severe and prolonged fevers (Parkes *On the Urine*, p. 243).

**TYPICAL RANGE OF TEMPERATURE IN A CASE OF PROTRACTED FEBRICULA** (*ephemera protracta*). THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wunderlich).

	1.	2.	3.	4.	5.	6.
	M	E	M	E	M	E
105						
104						
103						
102						
101						
100						
99						
98						

LINE OF NORMAL TEMPERATURE, 98° FAHR.

In cases of more protracted febricula, the early phenomena are similar to the shorter cases, and the protraction is mainly due to the slowness of the defervescence—an example of *lysis*. The phenomena of such defervescence, as indicated by the range of temperature, are shown in the foregoing diagram.

As a rule, these fevers are not serious; but the degree of reaction has always a relation to the state of the constitution, whether sthenic or not (MOREHEAD).

**Treatment of Febricula.**—Such means as emetics, purgatives, tepid sponging, diaphoretics, and antiphlogistic regimen, are to be employed. In plethoric individuals, where there is much headache and flushing of the face, a moderate general bloodletting, or leeches to the temples, may be expedient, but such remedies are not often necessary (MOREHEAD).

#### SIMPLE CONTINUED FEVER.

LATIN, *Febris continua simplex*; FRENCH, *Fèvre continue*; GERMAN, ———; ITALIAN, *Febbre continua semplice*.

There are not a few physicians who doubt the occurrence of *simple continued fever* as distinct from the continued fevers already de-



most importance to science, for more extended information regarding them will either connect them with forms of the fever between which they seem to stand; or these "doubtful cases" will eventually separate themselves into distinct forms, whose history is still unknown. In such doubtful cases observations regarding the correlation of temperature, the excretions, the succession of phenomena, and general course of the disease, are imperatively demanded.

The poisons of tropical fevers especially require to be carefully studied, and the phenomena of the febrile state which accompanies them embrace some medical problems of the most abstruse nature. Physiological data of an exact kind are now beginning to rise around us, which will give a standpoint for comparison in the study of the phenomena of fever in the tropics. Extremely important observations are being worked out by Dr. Emile Becher, of the Army Medical Department, regarding the influence of tropical climate on the excretions of the urine in relation to the body weight. At great personal sacrifice and denial of self, he has twice undertaken such investigations on his own person in voyages to India, round the Cape of Good Hope. With all such exact information, and the improved physical aids to investigation, it behooves the physician and pathologist to investigate medical problems with the same logical rigor and severity as a chemical or an astronomical theorem demands. On this important point the opinion of one may be especially quoted, whose experience as a teacher of clinical medicine has been great, and whose philosophical investigations into the nature of fever, in particular, command the respect of all. On this point Dr. Parkes thus writes: "The power of observation in medicine is a kind of tact, which ought to be cultivated with the same assiduity as the chemist practises when he learns how to manage his delicate manipulations, or the astronomer when he wields his wondrous tube. In medicine the observation and recording of phenomena have been held to be an easy and trifling task, which any tyro was competent to do. Hence half the error and uncertainty of medicine. Inaccurate, that is, erroneous and incomplete observation, has been the cause that, till within these few years, the fevers of cold countries have been so absolutely uncomprehended, and that the fevers of hot countries are still shrouded in obscurity. The most valuable addition any one could at present make to our knowledge of tropical fevers would be a simple record of all the cases in an epidemic. These cases should be observed with the keen tact of a Chomel, and recorded with the fidelity of a Louis. We want no explanation or word of comment added to them; we want merely the cases. Then, when the numbers are sufficient, we should certainly begin to put order into this chaos. And let not any one who may have the opportunities be deterred from the task by that fallacious, and, we beg to say, most reprehensible argument, with which some people may favor him,—viz., that his cases will be 'tedious,' 'heavy,' and 'unread.' Unread they will be, certainly, by some of the profession, who consider their routine practice as great an effort as their intellect will bear; but read and analyzed, we will venture to say,



plies itself by its passage through the human system, and which reproduces the same specific true yellow fever. The type of this fever is continuous. Pyrexia, delirium, suppression of urine, black vomit, are the leading symptoms of this fever—the *hæmagastric pestilence*, as it has been also called. (2.) That there are other fevers, and especially severe marsh fevers, in certain geographical limits, which have a close resemblance in symptoms to the contagious and specific yellow fever. So also, it is said, have fevers arising simply from a high temperature acting on an unseasoned subject. On this point my friend and colleague, Dr. Maclean, Professor of Military Medicine, who has had twenty-two years' experience of East Indian fevers, writes me as follows:

“I am now myself a firm convert to the doctrine that yellow fever is specifically distinct from remittent. To this opinion I have come with a full knowledge of the fact that some cases of remittent fever in India closely resemble some of the forms of yellow fever. But of this I am now certain, that the yellow fever of the true yellow fever zone is unknown in India where true malarial fevers abound. There is in true yellow fever, for the most part, an absence of that periodicity which is an unfailing characteristic of true malarial fevers. Then there is the difference so well insisted upon by Blair in true malarial fevers. Men do not pass from recovery to health, as is the case in such a marked degree in yellow fever, after which there is no, or very little, evidence of the existence of any cachexy. Malarial fevers exist and are destructive at a temperature at which yellow fever is at once destroyed. Albuminous urine is almost invariable in yellow fever—only occasional in remittent. There is in yellow fever an unexampled range of hemorrhages; in remittent fever these hemorrhages are often, indeed generally, absent. Quinine has a power over malarial fevers that is beyond the reach of doubt or cavil; the same is not true of yellow fever. Men suffer from malarial fevers again and again; second attacks of yellow fever are, to say the least, rare.”

The correctness of the above view of this important question has also received a remarkable illustration in some observations made in Mexico by the Medical Staff of the French Army serving there.

Yellow fever had disappeared from Vera Cruz. In the month of October, in the middle of a sudden augmentation of sickness, several severe cases of a disease like *vomito* appeared. The physicians dreaded a return of yellow fever in its epidemic form, in spite of the relative abatement of temperature and the almost constant prevalence of a northeast wind. These fears were augmented by the disembarkation at this time of a great many fresh troops; also by the suddenness of the invasion of the disease, showing itself in a great many cases by hepatic symptoms, bilious vomiting, fever, prostration, articular pains; the fever being continuous, or at least without sensible remissions, in many cases. But soon many circumstances demonstrated the groundlessness of the fear, and showed that the disease was malarious. These were,—(1.) The quickly recognized efficacy of quinine, which in the month of May, when true yellow fever raged, gave only negative results at the best; (2.) The rapid supervention of splenic enlargement, often attended with





yellow fevers of America, which are of marshy origin, black vomit is a usual symptom (Boott).

These two events—namely, yellowness of the skin and black vomit being of themselves insufficient as diagnostic marks of true or specific yellow fever, additional grounds of difference are found,—(1.) In the type of the fever, which is continuous and not remittent; (2.) In the fact that it occurs, as a rule, only once during life; (3.) In the fact that it is propagated by specific media from infected persons or places to others. But although in no one of these phenomena, taken singly, except in that of its communicability from person to person, do we find any definite characters to rely upon to prove the existence of a formal and specific yellow fever, yet, in the general assemblage and collocation of symptoms, peculiarities do present themselves which are easily discernible by an experienced eye.

Though the subject of tropical fevers is too little known to warrant decided opinions on many points, yet the true yellow fever, or hæmagastric pestilence, is now so clearly stamped with characters so peculiarly its own, that it takes its place as a specific fever of a continuous and generally rapidly fatal type. Its pathology is best exemplified in the history of such isolated outbreaks of it as are to be found in the cases of the "Hussar" (BLANE), the "Bann," the "Kent," the "Scout," the "Eclair," the "Hankey," the "Icarus," the Lisbon epidemic of 1857, and the importation of the disease from Havana into the port of St. Nazaire by the "Anne Marie" in 1861; and no description of yellow fever can be complete which does not give an account of some of these remarkable instances of this disease.

An analysis of all the circumstances connected with the "Eclair" shows (1.) That the immediate consequences of landing the crew at Boà Vista were a thorough intercourse with the inhabitants, and the communication to them of the same fever with which the "Eclair" was infested. For some time before the arrival of the "Eclair" it is certain that the island of Boà Vista was perfectly healthy; and this was true also of all the other islands of the group (ALMEIDA, MACWILLIAM). So great, also, was the dread of the disease among the inhabitants that the consul had great difficulty in procuring laborers; nevertheless, the crew managed to smuggle vast quantities of spirits, and, of course, it is possible that more secret intercourse went on than can be gathered from any official reports. Certain of the inhabitants were also brought more or less in contact with the crew of the "Eclair." There were—1st. The military guard at the fort; 2d. The laborers employed on board the "Eclair"—forty-one in number; 3d. The laborers employed in the launches, or at a coal-heap on a small island—forty-six in number; 4th. Washerwomen who washed the officers' clothes—seventeen in number. In addition to these, Captain Estcourt, the commander of the steamer, lived at the consul's house; the gun-room and ward-room officers and midshipmen occupied a house in Porto Sal Rey; and leave was given to the warrant officers and a few of the men, one of whom stopped in the town for two nights. The



period of latency for the variolous poison. Another case is cited—that of a boy, Lambert—which shows that the period of latency could not have been less than *eight* days. The experience of the Lisbon epidemic marks the time of incubation as varying from two to ten days, and in some instances extending to fifteen days. The importation of yellow fever by the ship “Anne Marie” into St. Nazaire—a town in the department of the Loire, about  $47^{\circ} 30'$  north latitude—in the summer of 1861 (*Ann. d'Hygiene*, Oct., 1863, p. 416), confirms the belief in a lengthened period of incubation; and from a careful analysis of the history of specific yellow fever cases, I think it will be found that the period of incubation tends to lengthen with the transportation and propagation of the disease into latitudes the most remote from the equator.

The history of the importation of the disease into St. Nazaire is as follows: About the 13th of June the “Anne Marie,” a wooden sailing vessel, laden with cases of sugar, left Havana, having been there a month during the prevalence of a severe epidemic of yellow fever. None of the sailors suffered so long as she lay at Havana, except from a little depression, loss of appetite, and a certain tendency to vomiting. After leaving Havana for France there was no sickness for seventeen days. On the 1st of July two sailors were attacked (without precursory symptoms) with violent shivering, pallor of the face, injection of the eyes, congested lips, and continued delirium. One died in twenty-three hours, the other in one hundred and ten. On the following seven days other persons were attacked, making in all nine cases out of a crew of sixteen persons. Only two deaths occurred. The ship arrived at St. Nazaire on the 25th of July with seven men still sick, but all of them convalescent, thirteen days having elapsed from the date of attack of the case last taken ill. Therefore, having had no deaths and no fresh cases for ten days, the “Anne Marie” was not placed in quarantine at the end of her voyage on arrival at St. Nazaire. Near her, as she lay in that port, there were anchored two ships of the imperial navy—namely, “Le Chastan” and “Le Cormorant,” the former touching her. Three other ships, “L'Orient,” “Les Dardanelles,” and “L'Arequipa,” were also near her. According to the custom of the port, the sailors of the “Anne Marie,” being only engaged for the voyage, quitted the vessel on her arrival, and were dispersed throughout the town. The commander, who had been ill, went home, the vessel was left to the second in command, and the places of the men were taken by seventeen fresh men, to discharge the cargo of sugar. These men were strong, very robust, and completed the discharge of the ship in eight days. Of these men twelve or thirteen were attacked with yellow fever, and many of them died.

“Le Chastan,” having been close alongside the “Anne Marie,” left on the 29th July, and sailed to Indret, on the Loire, 44 kilometres distant. Her crew, five in number, seemed in perfect health when they arrived at Indret; but on the 1st of August (*i. e.*, three days after leaving St. Nazaire) a man fell sick of yellow fever; then the remaining four by the 5th of August were all ill; and by the 10th of August all the five men, the crew of “Le Chastan,” were



ing in, as well as the inclosure of coal in tropical climates, as Mr. Macdonald notices, is worthy of the attention of the proper authorities.

All the cases, like the "Eclair," the "Bann," the "Imaum," the "Icarus," the "Anne Marie," the "La Plata," agreeing as they do in all their main features, it is impossible to doubt the existence of a multiplying infecting virus as the specific cause of yellow fever; and in cases where it was transported, imported, and propagated, the fever, except in certain cases, seems to have arisen only in persons who had been exposed to whatever deleterious influence was exercised by the atmosphere of ships in which cases of yellow fever existed. Moreover, the facts connected with the "Imaum," the "Icarus," and the "Barracouta," show that infected places and persons are alike dangerous to those who are at all susceptible; and in the cases of the "Imaum" and the "Anne Marie," it was shown that a disease taken in a certain locality, and spreading from person to person, may finally affect a second locality through their medium. It appears also to be quite a mistake to suppose, as Mr. Macdonald observes, that no individual can communicate the disease to another unless he himself is actually under its influence at the time; or, secondly, to consider such an individual as differing in any essential particular from an infected locality. Indeed, a ship itself is only an individual on a grander scale. This is the view Mr. Macdonald takes with regard to the "Imaum," receiving the disease from the officers of the "Icarus;" and it is also the view which explains the events which followed the contiguity of the ships to the "Anne Marie" in the harbor of St. Nazaire. To some extent the disease spread through personal intercourse to persons who were not near the ship,—in one very important case, that of M. Chaillon, a physician at Indret, who is said never to have been near the ship nor the town of St. Nazaire, but who contracted infection from four laborers who came infected from the ship, and whom he attended medically at their houses. He contracted yellow fever, and died. In a second case, one of the ship laborers, who himself had yellow fever, is said to have carried the infection certainly to his wife, and perhaps to an old man in whose house he and his wife lodged. All of them were attacked with yellow fever—the old man fatally. Unquestionably with regard to the "Anne Marie," and doubtless also with regard to the other ships, *the ships themselves*, irrespectively of sick persons in them, were *foci* of yellow fever infection. The men therefore, no doubt, carried infection passively, as they might have carried an odor from the ship, or as a student carries the smell of the dissecting-room on his clothes, especially felted textures. "I have often looked upon my own monkey-jacket with horror," writes Mr. Macdonald, "as the possible means of communicating so formidable a disease to others." Men thus laboring in the hold of infected ships, without themselves contracting yellow fever there, might carry infection to their homes, in climates and places where yellow fever may prevail. And it is a question how far, like cholera and typhoid fever *excreta*, the poison of yellow fever may not increase in places where decomposing animal material abounds.



imported from Havana by the bark *Adventure*, which put into Key West in distress, about the 20th June. It lay in quarantine thirteen days. Sixteen days after leaving Havana, the first and second mates fell ill with the disease, and in two days these men and two others of the crew, suffering from the fever, were taken on shore and placed in the Marine Hospital. July 27th, a soldier of the 90th New York was attacked with yellow fever, which subsequently spread through the garrison, there being 331 cases and 71 deaths.

The steamer *Delaware* with a detachment of the 7th New Hampshire, arrived at Hilton Head, S. C., from Key West, early in September, 1862, and, after a short quarantine, landed her passengers September 8th. Soon after several of these were taken ill with the fever, and eight died. On the 9th October a quartermaster's employee, living close by the quartermaster's depot in which a lot of tents brought by the *Delaware* had been stored, was attacked; subsequently, a number of officers, soldiers, and men, employed in the quartermaster's department, and all living around the storehouse in which had been put the tents brought from Key West by the *Delaware*, became affected. The hygienic conditions around this wharf were bad. The number of cases of the second outbreak was 30 and 17 deaths. The disease did not spread amongst the troops in garrison, nor did any of the physicians, attendants, or patients, suffering from other diseases in the general hospital, where the cases of yellow fever were treated without separation, contract the disorder.

In the same year (1862) there were outbreaks of the fever at Charleston, S. C., and at Wilmington, N. C.; and there is good reason to believe that it was introduced into both ports by blockade-runners from Nassau, N. P., where it prevailed.

Early in the autumn of 1864 yellow fever appeared at Newbern, N. C., and continued until the end of November; 705 cases and 288 deaths were reported amongst the white troops, and 38 cases and 15 deaths among the colored troops. Evidence is wanting to show the exotic origin of the disease, and it is claimed that it was of domestic generation, the local hygienic conditions being excessively bad. Previous to the outbreak at Newbern, the fever had appeared at Charleston, S. C., but there is no proof of any communication having been had between the two towns.

On the 10th October, 1864, the first case of yellow fever appeared at Wilmington, N. C. About the last of the previous August, two blockade-runners, with cases of the disease on board, were at quarantine three miles below the town. The quarantine was subsequently removed to near the mouth of Cape Fear River, and here, on October 1st, there were fourteen blockade-runners, and on all of them the mortality from yellow fever was very great. At this time the fever spread from the ships to the shore, the first cases being in the houses nearest the quarantine, and nearly one half of the inhabitants of the town died of it. Goods were known to have been smuggled on shore, and it is very likely some of them were carried to Wilmington.

During the autumn of 1864, 191 cases and 57 deaths occurred on board twenty-five naval vessels lying in the Mississippi River about and below New Orleans. It is claimed that these cases were spontaneously generated, and the bad hygienic condition of the iron-clads furnished all the alleged necessary factors. But the Spanish man-of-war *Pizarro*, with yellow fever on board, had been sent on the previous 4th of July to the quarantine, the first cases of the disease appearing on the 12th September. There were 12 cases and 3 deaths among the employees and guard at the Naval Hospital and the boat-landing at Erato Street; and five cases of





A rare manifestation of capillary irritation in yellow fever, consists in an efflorescence of the skin in the form of a subcutaneous rash on the chest, and extending over the abdomen and arms. Rose-colored spots, of a somewhat circular shape, have been noticed on fine, delicate, sensitive skins, varying from the size of a flea-bite to what might be covered with the point of the finger. They result generally from mosquito-wounds, and become hemorrhagic at the end of the disease when it terminates fatally.

*Bloody furuncles* appear late in the order of symptoms, and are to be regarded rather as sequelæ. Their most common site is on the wrist, over the metacarpal joints, along the front of the legs, below the scapulæ, and over the hip, in the parotid region, and over the forehead and lip. They are generally in close proximity to the smaller arterial branches, such as the ulnar, radial, anterior, tibial, gluteal, intercostal, and facial arteries. They become tender, acuminated, and inflamed, and sometimes form large abscesses of purulent matter, with a pale or inflamed surface, and this chiefly when below the scapula or over the hip. Generally on the legs they are flat, present no inflamed appearance, but show a flat purplish vesication, about the size of a split pea or a sixpence.

Two, three, or even four forms or types of yellow fever have been described by authors. These have been very clearly defined by my friend, Dr. Lyons, of Dublin, in the Lisbon epidemic of 1857, which he investigated with so much care and enthusiasm.

The *types, groups, or forms* which he found capable of clinical recognition are,—(1.) The algid form; (2.) The sthenic form; (3.) The hemorrhagic form; (4.) The purpuric form; (5.) The typhous form.

The first of these, namely, the algid form, is that which presents the most rapid course, and the earliest and greatest amount of prostration of the vital powers. These are the cases which are suddenly killed with the poison. "The patient, while in the enjoyment of his usual health, and in the midst of his usual occupation, feels suddenly the effects, as it were, of a sudden blow from a heavy bar on the back, falls down while walking (or if standing), and dies within a few hours in profound collapse, and after exhibiting more or less of the other symptoms of this fever." The countenance became sunken, the eye dull and filmy, the surface cold, and the patient felt cold, depressed, and wretched. The face became of a dirty livid hue, and this appearance extended to the trunk and limbs, the surface then presenting innumerable points of minute venous congestion, and sometimes purpuric spots and patches of various sizes. In extreme cases the lips, the breath, and tongue were cold, with a temperature in the axilla not more than  $96^{\circ}$ , the pulse being small, feeble, and quick; and when the cardiac action became feeble the radial pulse would be obliterated.

The *sthenic form* is a marked contrast to the algid. It is especially well marked in both sexes at the prime of life, and in persons with well-developed muscular frames. Such cases are characterized by well-marked febrile symptoms, severe and persistent headache, much rachialgia at the outset, a high, full, and hard pulse, occasionally thrilling and resisting, with flushed face and throbbing



considerable strength, though still compressible, and may vary in these respects within certain limits until it becomes feeble. In those cases which have proved rapidly fatal there has been a marked gradual decrease in its strength; and finally, when the ferrety eye grows clear, and a pallor of countenance shows the mischief of blood exudation going on within, it is scarcely to be felt.

Observations made on the urine, in yellow fever, by Dr. Blair, show that it is always acid in the first stage, and continues so generally till convalescence, when it becomes alkaline, or until it becomes heavily charged with bile. During the early stage the urine is normal in color, clearness, and quantity. About the third day the color alters, and becomes of a sulphur, primrose, straw or light gamboge hue, perhaps slightly turbid, and with a little floating sediment. The color deepens till it becomes yellow or orange; and if the case ends in convalescence, the urine is copious, and may appear black. Sometimes the urine has a pale, watery, smoky appearance, with a layer of blood-corpuscles in the sediment, and sometimes it is very bloody.

Albumen appears on the second or third day generally; in some cases as early as the first day; and in a few cases not till the day of death, and after black vomit has set in. Albumen appeared in every fatal case of normal duration. It sometimes ceased suddenly in convalescence, and always before the yellow suffusion of skin and eye, or bile in the urine, had disappeared. Between the eleventh and twentieth day of grave cases it generally disappeared. Its color was never white. When the urine appeared turbid, it was due to the presence of mucous epithelial matter, coagulated albumen, coats of the urinary tubuli, or fine capillaries of the kidney or mucous membrane passed out with the urine. The tube-casts are generally short, thick, club-shaped, and opaque, attended with large organic cells and epithelial scales. Crystalline deposits are rare. In females the catamenia are sure to appear, whether due or not. No sign is so dooming as a suppression of urine, *black vomit* not excepted. The alvine evacuations may be black towards the close of the disease, or very dark green, and bilious; but after the black stools have ceased, they are succeeded by evacuations which resemble fine, dark, sandy mud, and named the "caddy stool." As the disease still further advanced, and towards its fatal termination, the dejections again changed their character. They became scanty and mucous, of various consistence and color. These mucous stools almost always appeared *after* black vomit, and were contemporaneous with the scanty urine before described. The alvine evacuations in yellow fever, from the beginning to the end of the attack, are always alkaline, except in one instance, that of the black vomit stool; in that it is always acid. Its chemical quality is evidently due to the admixture of a portion of the black vomit, which has descended (if not found in the intestines) by peristaltic motion into the intestines, and mixed with the scanty mucous stool, and in such quantity as not only to neutralize it, but to be in excess. The scanty thick mucous stool—almost a jelly—has generally a little thin serum around it in the bottom of the pot. The bulk of all these varieties of the scanty



**Prognosis.**—The data to judge from may be arranged into symptoms which are favorable, and those which are not so. (1.) *The favorable symptoms are*—A slow pulse and moderate temperature of the body, and quiet stomach. Streaks of blood during the stage of black vomit, or after acid elimination has set in, are favorable, if the corpuscles are found entire. If the urinary secretion continue, and the black vomit be scanty from the first, or is afterwards suppressed, the patient may yet survive. Urine simply albuminous is a less serious sign than when it also contains tube-casts. Free, copious urine, no matter how dark or bilious, is the most favorable of any single sign. Prognostics may be derived from the effects of treatment. (2.) *The unfavorable signs are*—The more fiery crimson the tip and edge of the tongue, the more irritable the stomach, the severer the headache, the worse the prognosis of the first stage, and *vice versâ*; but a streak of blood in the early vomit indicates much danger from the attack. In the second stage the earlier or more complete the suppression of urine, and the more copious the ejections of black vomit, the more imminent the danger. If the urine be scanty, and loaded with tube-casts, entangled in epithelial and coagulable matter, the light buff-colored curdy sediment before mentioned indicates a complex lesion of the secreting structure of the kidney. It is the urine symptom in its maximum of severity, and is as fatal as if the suppression had already occurred. Blood-corpuscles in the urine are not to be looked on with apprehension. A faltering of the articulation is a bad prognostic, and a difficulty of protruding the tongue enhances it. The danger of the case is enhanced by inflammatory complications, and by hypertrophy of the heart. A recent residence in a temperate climate; the *race* or complexion of the individual; the fact of his previously having suffered from an attack, will enter into an estimate of his chances of recovery.

Prognosis is declared by Robert Jackson to be treacherous and difficult in the extreme.

**Treatment.**—An early attention to first symptoms among the susceptible is of the greatest value in saving human life.

The diagnosis of cases in which the attack has been said to have been “aborted” by remedies may be questioned, and such a belief is opposed to the doctrines of sound pathology. Moreover, the “heroic” doses of calomel which were given in such cases, combined with quinine, cannot be too strongly discountenanced, for “they were first recommended on the strength of a crazy hypothesis” alone. The practice is said to have frequently proved successful in Jamaica; but, according to Dr. Davy, it was not attended with beneficial results at Barbadoes; and the American physicians at New Orleans have not found it to answer their expectations in stopping the fever.

The large and frequently repeated doses of quinine were often also highly injurious (LAWSON). This discrepancy may in some measure be explained by what has been stated at the outset in explaining the pathology of this peculiar fever. It is in cases where the fever is of the periodic or paludal form, and not the continuous or true yellow fever, that quinine may be of use, if the system



auxiliary means of relieving the blood of its poisonous, metamorphosed, and effete constituents, the onus of which is now thrown on such vital organs as the stomach and lungs. At one time the heat of the surface was so ardent and persistent that the wet sheet failed to reduce it effectually."

The most distressing symptom in yellow fever, both to the patient and the medical attendant, is irritability of the stomach; it is so constantly present, and so often uncontrollable, that the knowledge of every available means of checking it is of the utmost importance.

The *food* during the course of yellow fever should be of the blandest description—chicken tea, arrow-root, sago, and barley-water constituting the chief articles; and these should be taken in minute quantities at a time when the stomach is at all irritable. This rule applies to drinks of all kinds. The patient is greedy for a large draught of fluids; but by sucking them through a glass tube, or a straw, or a hollow reed of small bore, or by the tea- or tablespoonful, they are much more likely to be retained. A cold infusion of oatmeal was found an agreeable drink for Scotch seamen, of which they did not seem to tire. A dislike of sweets was observed among the patients; and when lemonade was asked for, the usual quantity of sugar was objected to, probably from its rendering the liquid too dense for ready absorption by the stomach, and therefore less quenching. *Tea* was found so uniformly to disagree with the patients, and cause vomiting, particularly in the advanced stages, that at length it had to be expunged from the yellow fever dietary. Dilute alcoholic drinks were given freely, and with good effect. Where brandy could be obtained pure (tolerably free from acidity and fusel oil), and was well diluted with water, that spirit answered every indication. Sometimes the effervescing wines were relished and retained, but they are very liable to the objections of containing foreign matters and the products of mismanaged fermentation (BLAIR).

Mr. John Denis Macdonald has several times proved that four or five minims of chloroform prepares the stomach for the reception and retention of food, by lessening its irritability, and the dose should be repeated a short time before food is again taken, as the effect of the chloroform is transitory. On the same principle the administration of *chlorodyne*\* may be advocated. Lime-water has been also found

---

\* The composition of *chlorodyne* is variously given by chemists, but the following formula may be given as a very useful one:

R. Chloroform, fʒiv; Æth. Sulph., fʒii; Theriacæ, fʒi; Mucilag. Acaciæ, fʒi; Morph. Muriat., gr. viii; Acid. Hydrocyanic dil. (2 per cent.), fʒii; Ol. Menth. pip., ℥iv, ad vi; *misce bene*.

Syrup or water may be added to the mucilage of gum, and tincture of cannabis Indica (5 to 20 minims), or other anodynes, may be added at the time of prescribing, if deemed desirable. The difficulty in compounding *chlorodyne* is in getting the chloroform to mix with the treacle. It will not do so alone, but the use of a little thin gum, or even water, effects their mixture almost at once. The morphia ought first to be dissolved in the chloroform; then mix with the treacle the gum or water, first using about the same bulk of chloroform and treacle, afterwards adding the rest of the chloroform by degrees, constantly shaking briskly the bottle in which it is made up;





ally and in unusual force in the delta of the Mississippi during this period of immunity from that disease" (p. 253). Previous to the war the annual average of yellow fever victims in New Orleans was about 1000. The average annual death-rate, from all causes, from 1829 to 1861 was six and a half per cent. There were years when the death-rate exceeded ten per cent. During the aforementioned years, the town was full of unacclimated persons. "One hundred thousand Northern men annually arrived in or passed through, without a single individual being smitten with yellow fever" (HARRIS, *l. c.*, p. 256). These summers passed without a sign of yellow fever epidemic. Besides the observance of a rigid quarantine, the strictest sanitary regulations were enforced by the military government, and to them is to be ascribed the exemption. The thorough cleansing of the towns of Savannah and Charleston, after their occupation by the U. S. troops, and the rigorous sanitary regulations maintained, together with a *military* quarantine, have kept them, it is believed, from any outbreak of yellow fever since 1864.

In the Army Report, already quoted from, Dr. Woodward holds this language: "In conclusion, a few words may be said with regard to the relations of the facts set forth in this report, and in the appended documents, to the prevention of yellow fever. It is to be regretted that the experience of the army throws no more satisfactory light on the treatment of the disease, but it must be admitted that it is most instructive with regard to measures of prevention. Besides those general hygienic precautions which are so important in the prevention or mitigation of all epidemic diseases, two simple and effective measures would appear to be specially indicated by the experience of the army during the war and subsequently. The first is quarantine, as a means of preventing the introduction of the disease; the second is the prompt movement of the command to some rural site on the appearance of the fever among the citizens of the town at which it is stationed, or even after the disease has appeared among the men of the command itself.

"With regard to quarantine, it is well known that a great difference of opinion exists among civil physicians; nor is this surprising, since, in populous cities, approached by many routes of travel, a foreign disease may readily be imported by persons eluding an imperfect quarantine. In such a case it may be quite impossible for the physicians of the place to determine the circumstances, naturally concealed by those who have broken the laws or regulations on the subject.

"In the case of military detachments, however, especially during times of peace, the movements of individuals being so much more readily known, the mode in which such diseases are introduced can very generally be recognized; and hence it is not surprising that recent distinguished English writers on subjects connected with military medicine—Dr. Aitken, in his *Practice of Medicine*, and Dr. Parkes, in his *Hygiene*—are advocates of the doctrine of importation. Dr. Parkes expresses the opinion that the incubative period is longer than is usually supposed, probably often fourteen or sixteen days. Several facts set forth in the appended documents would seem to show that, in certain cases, the disease may be delayed as long as three weeks after exposure. The minimum period of an effective quarantine against yellow fever may then be set down at about twenty days. Twenty-five or thirty days would be better if attainable.

"Should the disease, unhappily, be introduced through neglect to provide an efficient quarantine, it becomes the imperative duty of the medical officer to recommend the immediate removal of the command to some



tive or pernicious form of the disease. During the year ending June 30, 1862, more than one-fourth of the army suffered from these affections. For the same period, there were 123,763 cases of remittent fever, and 1537 deaths. Malarial fevers prevailed to a great extent amongst the Confederate troops, and, in certain localities, the efficiency of entire commands was greatly impaired. In an average command of 878 men, stationed about the forts near Savannah, nearly one-half, on an average, were entered on the sick list each month, and the new cases per month was 220. Throughout a period of fifteen months more than one-fourth the command was unfit for duty, and during the autumn months more than one-half the garrison was on an average incapable of performing military duty. During a period of nineteen months, January, 1862, to July, 1863, inclusive, there were reported in the Confederate army serving along the coast of South Carolina, Georgia, and Florida, with a mean strength of 25,723 officers and men, 157,013 cases of disease and wounds, and of this number 41,526 cases were recorded under the head of some form of malarial fever—a little more than one-sixth, or 16.3 per cent. of all the sickness. The per cent. of malarial disease in the commands was 161, or each man on an average had been entered upon the sick list with paludal fever 1.6 times. In the Confederate forces serving along the coast of the Gulf of Mexico, each man on an average was entered on the sick list, with one or another form of malarial fever, a little over twice in eighteen months. In the (Southern) army of Tennessee, the monthly ratio of cases of malarial fever to the mean strength of officers and men ranged from 4.2 to 17.9 per cent. Even the army of Stonewall Jackson, serving in the Valley of Virginia, an elevated and healthy region, suffered greatly from malarial fevers; for, during a period of ten months, with an average mean strength of 15,582 officers and men, out of 53,198 cases of disease and wounds entered on the field reports, 3876 are recorded under the heads of congestive, intermittent, and remittent fever (J. JONES, *U. S. Sanitary Commission Memoirs*, 1868).]

**Pathology.**—In these forms of fever a malarial poison of an unknown kind, generated in paludal regions or littoral districts, is absorbed, and affects the blood, as cholera, typhus, and other miasmatic poisons do. The poison, in the absence of any better name, is known as "*malaria*;" and as physicians have merely inferred the existence of such a poison, no exact knowledge has yet been obtained as to its nature and source. Indeed, it still remains to be shown that *malaria* have a substantial existence. No poisonous principle has yet been chemically demonstrated in the air of malarious regions. But many other acknowledged disease poisons are in a similar predicament as to proofs of their substantial existence; and the general impression with regard to *malaria* is, that it is presumed to exist as a gaseous fluid in the atmosphere of certain regions.

After a period of latency, more or less long, functional disorders of the great nervous centres are brought about, terminating in the phenomena either of intermitting, remitting, or yellow fever. These fevers may exist without any alteration of structure being set up, and the patient often dies from the severest forms, with hardly a trace of disease being discoverable. In the milder forms of these fevers, however, a greater number of organs and tissues are morbidly altered than perhaps in any other disease, as the liver,



The functions of the peritoneum may be alone deranged, so as to produce dropsy; but every form of peritoneal inflammation may precede or accompany the *ascites*,—as the serous or the purulent, with diffuse or partial local adhesions.

These are the most usual alterations of function and of structure in the mild paludal fevers of the present day; and in estimating the relative frequency of these secondary affections, *ascites* is the most common, then *jaundice*; while *peritonitis*, *hepatitis*, and *splenitis* are less frequent, and occur, perhaps, in nearly equal proportions.

The pathological phenomena which attend severe intermittent and remittent fever are much more severe, and extend over a greater number of organs. The information afforded us by the dissections of Davis, and the observations of Sir Gilbert Blane, in the cases of the Walcheren remittent; of Jackson in those of the West Indies; of Burnett in the Mediterranean, enable us to understand at least the tendency of the morbid action. Sir Gilbert Blane, in his observations on the Walcheren fever, remarks that the structural derangements were more frequent (especially swelling of the liver and spleen), which then occurred in a very few weeks. Such results seldom occur in England, except under a long continuance of the disease, or after frequent relapses. The morbid changes also extended to the mucous membrane of the stomach, which, in a few instances, was inflamed and ulcerated, and the ulcers had generally a sharp perpendicular edge, as if made with a punch. In cases which died dysenteric, the large intestines, and more particularly the sigmoid flexure and the rectum, were always much contracted, thickened, inflamed, and ulcerated; the ulcers being often so numerous and so confluent that the whole inner surface of the gut appeared in a state of granulation. There is a marked tendency in the phenomena of these paludal fevers to become inflammatory, the congestion of some organ proceeding at once to exudation from the blood-vessels into its parenchyma, which appears to be the cause of prostration and of fatal results. "The significant term *bilious*," writes Sir Ranald Martin, "as applied to these fevers of the East is not an accidental or a misapplied term, as modern statistics fully show. A severe disturbance of the hepatic function is almost universal in the progress of the remittent fevers in the East."

There is another remarkable tendency to be noticed in the persistent effects produced by intermittents—namely, that they impress a character of periodicity to subsequent ailments, especially neuralgic affections; and the disposition to the recurrence of these diseases seems to last for life. Susceptibility to the action of the paludal poison does not diminish, but rather increases by continued residence where it prevails. The returns published by the War Office and Army Medical Department show such a result in the West Indies. Thus, while the annual mortality among the troops resident one year in Jamaica was 77 per 1000 mean strength, in those resident two years it was 87 per 1000, while of those still longer resident it was no less than 93 per 1000.

"In making calculations of efficient force," writes Sir James Macgrigor, "this description of men could not be relied on for ope-



with certainty that they are cases of remittent rather than of yellow fever, or of yellow fever and not remittent.

[**Chronic Malarial Toxæmia** was very common amongst the United States troops in the late war, who were exposed to the influence of paludal poison. The manifestations of the poison upon the system are slow but characteristic. As observed in the army they have been thus described: The man is evidently out of health, and unfit for duty. He is said to be laboring under "general debility." There is a gradual loss of power, and fatigue comes on from slight exertion, with breathlessness and palpitation; the senses are dull and perverted; there are moroseness, despondency, and irritability; headache, and neuralgic pains in the course of the fifth pair of nerves; lameness of the muscles of the back and legs is often complained of after little exertion; occasionally, there is more or less diminution of sensibility or motion of the lower extremities, which become enlarged, and the integument is shining, smooth, and pits upon pressure; the appetite is capricious and lessened, and there is constipation, alternating with diarrhœa; the urine, at first copious, soon diminishes, with an increase of the urates and phosphates, and frequently of the oxalate of lime, and is loaded with epithelium; the bladder is irritable, with frequent micturition; the skin is harsh, dry, of a greenish-yellow hue and bronzed in portions; and the hair has a dead look and feel. Persons in this condition are very liable to acute disorders, particularly pneumonia, and which constantly are fatal.

**Morbid Anatomy of Malarial Toxæmia.**—The integument is bronzed, especially, in the regions of the face, neck, sub-axilla, arm, forearm, and outer side of thigh. This change consists in a pigmentary deposit resembling that of Addison's disease, and is to be distinguished from icterus, or the icteroid hue, which is probably from altered hæmatine. Leanness does not amount to emaciation; the fat has largely disappeared, but the muscles retain their fulness. The muscular tissue is generally of reddish-brown color, tears more easily than in its healthy state, but its specific gravity is not lowered. The blood is fluid, but fibrinous coagula full of white corpuscles are found in the cavities of the heart and in the great vessels. The chief and most characteristic changes are, however, to be found in the liver, spleen, kidneys, the lymphatic glands, and the intestinal glandular apparatus. The liver is large, of a pale reddish-slate or fawn-color, and its relative and absolute gravity are increased. It is firm to the touch and divides firmly; the faces of the divided parts are smooth and the edges sharp; the acini are small and indistinct, and the interlobular substance is increased in thickness and development. This increase of the interlobular substance is either by development of its own substance, or, as is more probable, by the addition of new material, albuminous in character. This encroaches upon the vessels, or deposits take place in the walls of the vessels, lessening their calibre and diminishing the supply of blood to the lobules; hence the hepatic cells become pale and shrink, and fatty transformation finally occurs. This fatty metamorphosis I observed but in a single instance; the liver was small and very flaccid. The secretion of bile does not cease; the gall-bladder is well distended, but the character of the bile is changed; it is usually dark brown and tarry in consistence. Corresponding changes occur in the kidneys. They are enlarged, their relative and absolute gravity being increased. They divide firmly. The cortical substance is whitish or fawn, and the cones of Malpighi are congested, purplish, and the papillæ red. The tubules are seen to be crowded with epithelial cells, and their walls





from a mere dot up to grains many times as large as red blood-globules. Frerichs states that some of the masses are  $\frac{1}{100}$  of a line in breadth and  $\frac{1}{20}$  of a line in length; and these he regards as probable casts of the smaller vessels. My own measurements correspond closely with these. I have not recorded any isolated grains of more than  $\frac{1}{200}$  of an inch in breadth, but have frequently observed aggregations of granules and "larger masses" which measured  $\frac{1}{800}$  to  $\frac{1}{700}$  of an inch;  $\frac{1}{200}$  to  $\frac{1}{150}$  of an inch was an approximate average size of the separate grains. Now when we bear in mind that the common width of red blood-corpuscles is in man about  $\frac{1}{300}$  of a line, and of the white corpuscles  $\frac{1}{250}$  of a line, we see that many pigment grains would be necessarily arrested where these could pass. And in different structures of the body the capillaries range only from  $\frac{1}{800}$  to  $\frac{1}{700}$  of a line in diameter.\* In *shape* these pigment-grains are very irregular, rounded, or sharp, with an angular, brittle-looking outline. Indeed the sharpness of their angles, which, however, are in no degree definite or uniform, "has suggested the term *crystalloid* as applicable to them (Lebert), or rather to the pigment-material common in the lungs."† And I may here allude to their close resemblance to the black matter so frequently seen in old coagula; in patches of chronic inflammation and ulcers of the intestines; and in the fluids vomited in cases of gastric cancer with a bleeding surface. Some writers go so far as to claim an identity,‡ but this is certainly premature. We cannot safely assert more than their common origin in some blood metamorphosis. In color the pigment varies almost as much as in size and shape. Usually it is deep black, quite opaque, and with abrupt, non-translucent margins. Then, again, we find a brown rim through which some light passes. Frequently the color is reddish-brown, or even reddish-yellow, as seen by transmitted light, and the granular scales are seldom opaque. In the splenic pulp I satisfied myself of the existence of red blood-globules in different stages, not only of disintegration, but also of advancing metamorphosis toward black pigment, and am therefore prepared to agree with Frerichs, "that these different colors represent the various stages in the transformation of the red pigment of the blood into melanotic matter." I have occasionally seen tinged gelatinous particles, as though there was a union between the coloring matter and some protein element. But of their intimate chemical nature we are ignorant. All agree, however, that while the pure black forms resist the action of even the strongest acids and alkalies, the paler products lose their color with greater or less rapidity under the influence of these reagents. (MEIGS, *l. c.*, p. 106.)

That the pigment-granules and masses are not foreign bodies, but have their origin in some abnormal action within the organism, and are the product of certain changes of the blood-elements, is probably certain. Whether strictly pathognomonic or not of malarial toxæmia, there is abundant evidence to show their constant connection with that condition, whether acute or chronic. In 90 cases of malarial fever, examined by Dr. Meigs, in 1865, in one only did he fail to discover, in the blood taken from the body during life, pigment-granules. Dr. Meigs gives the following conclusions:

1. That in examining blood during life with a view to determine the presence or absence of pigment-matter, great care is necessary to exclude

[\* Human Anatomy, Leidy, Philadelphia, 1861, pp. 337, 339.]

[† Clark, *op. cit.*, p. 611.]

[‡ It is the *pseudo-melanose* or *éléments hématiques* of Lebert; when crystalline the *hæmatoidin* of Virchow.—CLARK.]



in perspiration. At the end of the seventh day he ceased to bathe, but was, nevertheless, nightly, about the same hour, attacked with a regular intermittent paroxysm, consisting of the cold, hot, and sweating stages, which returned for about a week, when it ceased spontaneously on the occurrence of an event which kept him out of his bed at the hour of paroxysm, and induced him to take a ride on horseback, which excited and warmed him." Cases having their origin in such causes, however, are of exceeding rare occurrence, so far as the records of medicine show.

The concurrence of circumstances under which paludal and littoral fevers have been observed to become developed may be shortly stated as follows: (1.) A certain degree of heat. A high temperature is especially favorable to the production of malaria, and the more so when acting on moist alluvial soil. (2.) A certain relation as to season, variable with the geography of the locality in which such fevers prevail. The season of the year most marked in tropical climates is that which immediately succeeds the cessation of the rains, or, as it is called, "the drying up of the rains." (3.) Low swampy grounds and extensive rice-fields are well-known sources of malaria. In such districts clouds of mist are often seen, wafted along the earth's surface for miles; and it is believed that malaria, whatever be their nature, cling to such mists. But although it has been observed that absolute marshes do not always produce agues, nor that agues are always due to obvious marshes, yet it is generally found that in districts where such paludal fevers abound the surface is porous, penetrable, and retentive of moisture, although it does not appear on the surface of the ground; that the district had been at one time submerged; and that it continued slowly but constantly to undergo the process of desiccation: or while at certain seasons it imbibes moisture from local or meteorological sources, at other seasons it undergoes the drying process under intense solar heat. Such are some of the most sickly and febriferous districts in Europe, India, and America. For example, the Maremma of Italy; the district of the Lakes near Varna, in Bulgaria; many districts in Burmah; many newly cleared tracts in North America; and many parts in the south of Spain. In most of these places the conditions of the surface of the ground are very much alike. While no obvious appearance of a marsh exists, the vigor of vegetation is extreme, amphibious animals abound, of the batrachian kind, plants and cephalopodous mollusca of notoriously marshy regions find a habitat, and the rich alluvial soil is so imperfectly cultivated that the process of vegetation is not adequately exhausted, and a surface of humid ground is exposed to the solar heat, and so exhales a material which exercises a persistent deleterious influence on the human frame. It is believed that the number of insects and some reptiles with which a place abounds are more significant of its insalubrity than almost any other circumstance; and that a mixture of animal and vegetable matters undergoing decay give rise to miasms much more noxious than those resulting from vegetable matter alone. Dr. Fergusson, in *The Edinburgh Philosophical Transactions*, vol. ix, p. 278, was the first author who clearly proved that the drying of all



come from marshy districts, either quite recently or within a few months. The malarious influence, still in the metropolis, seems, however, sufficiently powerful to imprint a periodic character upon various local affections, and occasionally to give rise to fevers of a remittent type. Recently (in 1856) such affections have been unusually prevalent; but the forms of ague now met with in London are more tractable and milder than those which formerly prevailed (Dr. PEACOCK).

It is observed that the surface of the earth may be dried either by the direct rays of the sun, or by currents of hot dry air wafted over it, or by both combined; but it is principally by the direct rays of the sun that the deleterious material of the soil is liberated; and it seems to be at a certain period of this "drying up" process that the exhalations are more potent than at another time in developing paludal fevers. The exposed grounds, after clearing off the copious vegetation from dense jungles, so as to admit the influence of the sun's rays in "drying up," is known to be a fertile source of malaria.

There appears also to be a certain state of the human frame which renders it more than usually susceptible to this disease. The natives of warm and tropical climates are much less frequently and less violently attacked with paludal or littoral fevers than settlers or visitors from other lands, such as the natives of Europe or the northern parts of America. In the Mediterranean, along the coast of Africa, in the East Indies, in the West India Islands, in the Southern States of the Union, new-comers from the northern latitudes are almost invariably attacked, and suffer much more severely from the fever than those who have been long in the country. It has been also noticed that those who, after residing in a territory where paludal fevers abound, have been out of it for some time, an augmented susceptibility to renewed attacks of the fever becomes manifest on their return (CRAIGIE).

Other causes predispose to those fevers, and none more than laborious or fatiguing duty in military or naval operations, laboring in the sun, excess in eating or drinking, intellectual exertion combined with bodily fatigue, and a crowded state of the population. Indeed, *sunstroke*, or heat apoplexy, is regarded by many as a form of remittent fever (JOHNSTON, MARTIN, HILL).

When a remittent fever, or other paludal or littoral fever, has, under certain concurrent circumstances of weather, season, and physical peculiarities, made its appearance in any locality, it necessarily attacks all those who are by constitution, habit, and age, susceptible and predisposed; and the majority of these, especially if enfeebled by previous dynamic or organic disease, it destroys. The population, therefore, which outlives such an epidemic visitation are no longer equally susceptible, and are greatly less likely to be attacked the ensuing season, unless it is more febriferous than the past, which, though sometimes, is not generally the case. The effect of this, therefore, is, that while the endemial disease continues for a season to attack and destroy its *ordinary* annual proportion of the population, it does not for several years attack the *extraordinary* proportion, because that proportion is not yet ready for, or susceptible of,



**Symptoms.**—The disease may be sudden in its attack, and without previous illness; but more commonly it is preceded by general indisposition, headache, weariness, pain in the limbs, thirst, loss of appetite, white tongue and frequent pulse, high-colored urine and dark-colored discharge from the bowels. These *prodromes* are accompanied with well-marked exacerbations and remissions of fever, displaying a periodic tendency. After this feverish state has lasted from four days to a fortnight, the patient is seized with severe rigor, and the ague is manifested. The phenomena of an attack or “fit of the ague” are the following:

The paroxysm, like the disease, may be of sudden invasion, and the patient may be in good health up to the time of attack; or it may be preceded by languor, debility, frequent yawnings, and great unwillingness to make the least exertion. In whichever way the cold stage begins, the patient experiences first a sensation of coldness of the extremities, then of the back, and lastly of the whole body; at the same time the nails turn blue, and the features shrink, becoming pale and sharp. If the case be severe, the whole body shrivels up, turns purple, and the surface of the skin assumes that rough condition popularly named “goose-skinned.” The coldness increasing, the motor nerves of the fifth pair are affected, and the teeth begin to chatter; and this tremor extends to every muscle, till the whole body shakes with rigor. Cough, dyspnœa and oppression of the præcordia now occur, with a painful sensation round the temples and down the back. The patient often suffers from nausea and vomiting, and the latter symptom is speedily followed by the hot stage.

[The chill is sometimes so slight as to amount to only a feeling of chilliness along the spine, or over the extremities, or it may be limited to a single limb. The cold stage may be manifested merely by severe pain in the supra-orbital nerve, or there may be simply a drowsiness with excessive yawning or a lethargic state, preceded or accompanied with nausea and vomiting. Dr. Flint has known a state of intense nervousness take the place of the cold stage.]

When the cold stage has lasted a period varying perhaps from half an hour to two hours and a half, a reaction takes place, accompanied by partial warmth, or flushings. These extend, and at length the whole body acquires a heat greater than natural, or from  $105^{\circ}$  to  $107^{\circ}$ . As the heat returns, so also does the color, and the body, especially the face, becomes preternaturally swollen and red. The hot stage being formed, the heart and arteries beat with unusual violence, and headache, with a frequent full pulse, and all the distressing symptoms of continued fever, are present. “The mean duration of this stage is from three to eight hours. At its close a gentle moisture breaks out, first on the forehead, and thence extends till the patient lies in a general sweat, sometimes so profuse as to soak the bed and linen as completely as if they had been dipped in water. After the sweat has continued to flow for some time the fever gradually abates, a state of apyrexia ensues, the paroxysm is terminated, and (a sense of exhaustion excepted) the patient feels





*quotidian* there are two daily paroxysms. The *double tertian* is where there are two paroxysms and two intermissions in the forty-eight hours—the alternate paroxysms being similar, while those immediately following one another are not so. In the *triple tertian* there are two paroxysms on the odd, and one on the succeeding days. The true paroxysm in the *double quartan* takes place on one day, a slighter one on the second, while the third is a day of intermission, and there is another paroxysm on the fourth day resembling that of the first, and so on, in succession. In the *triple quartan* there is a daily paroxysm, but it varies on the first, second, and third days,—the paroxysms happening on the first and fourth, on the second and fifth, on the third and sixth, and so on successively, being respectively similar. In the *duplicated quartan* two paroxysms occur on the first day, while there is an intermission during the second and third.

A severe and dangerous variety of intermittent fever is the *congestive*, *pernicious*, or *malignant* intermittent, more frequently met with in the Southern and Western States, and occasionally in those parts of the Northern States where paludal fevers are endemic. During the first two years of the late war, there were reported in our armies 6081 cases of congestive fever, and 1381 deaths, whilst in 156,726 cases of all other varieties of intermittent fever there were only 407 deaths. There are two forms, the *comatose* and *algid*. In the comatose the head-symptoms may vary from lethargy to deep coma; the pulse is large, soft, and generally slow; the respiration is laborious, noisy, and infrequent; the patient lies on his back, unconscious, and cannot be roused; the limbs seem paralyzed; the jaws are locked; deglutition is difficult; there are, sometimes, epileptiform spasms, or active delirium. After the continuance of these symptoms for a variable time, sweating comes on, and there is gradual awakening, with an astonished look, and the senses are regained one by one. The *algid* variety is marked by an icy coldness of the surface, like marble or the collapsed stage of cholera. The extremities, face, and trunk, become cold in succession, the skin of the abdomen remaining warm longest; the tongue is pale, shrunken, and cold; the breath is chilled, and the lips livid. The action of the heart is feeble, and the pulse rapid, small, and almost extinct. The respiration is quickened, broken, and embarrassed. The mind is often undisturbed, and a sensation of repose is felt. The eyes are hollow, glassy, and surrounded with a bluish circle; the face is pinched, and all expression gone. If death does not happen in the fit, the pulse is slowly developed, and the heat of the surface gradually comes back, beginning at the abdomen and extending to the extremities.

The *congestive* variety of intermittent fever is very insidious, and may come on in the second or third paroxysm of a *quotidian*, or *tertian*; or it may be initial, though generally preceded by prodromic phenomena, as drowsiness, headache, languor, and gastric derangement. The accession of the *algid* form is sometimes sudden, the patient becoming rapidly cold, lying down, and dying in a few hours. If there should be more than one fit, each succeeding one is more severe. The patient may die in from two to twelve hours.]

Of these primary types it is believed that in Europe the *tertian* is by far the most common type, then the *quartan*, and lastly the *quotidian* (WATSON, COPLAND, CHRISTISON). But this law is by no means general; for M. Maillot treated 2354 cases of intermittent fever occurring in the French army in occupation of a portion of the northern shores of Africa, and he found of that number 1582



a whole day free of fever between every two days of the paroxysm. All the types of the fever present this characteristic peculiarity of a sudden and speedy rise of temperature to a high degree (mostly up to 105° or 106.3° Fahr.); and of an equally rapid and complete defervescence, till the period of another fever paroxysm comes about. This comportment as to temperature secures correctness of diagnosis in cases which may be obscure or ambiguous.

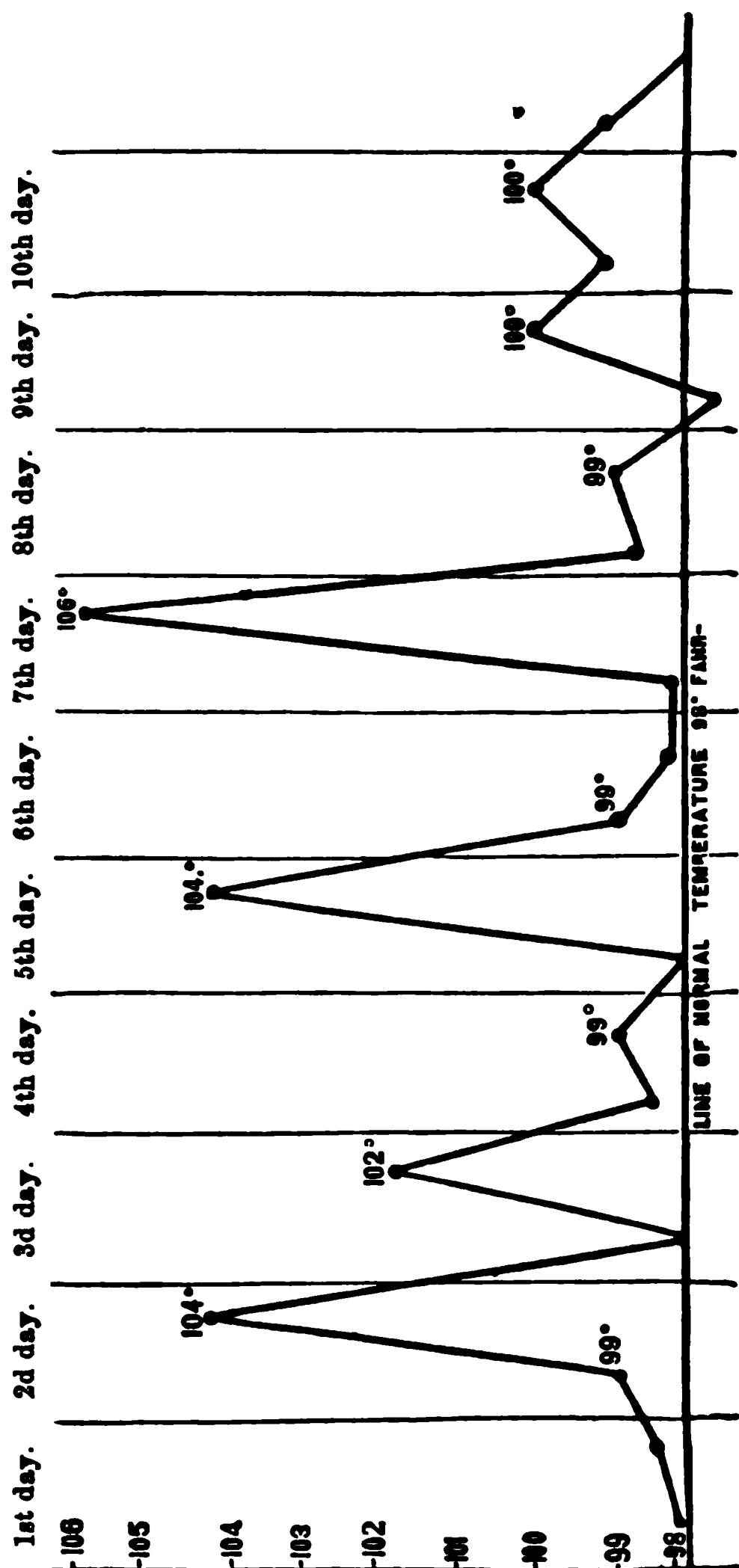
The annexed diagram and that on page 474 represent variations of temperature in cases of malarious fever.

(1.) TYPICAL RANGE OF TEMPERATURE IN A CASE OF INTERMITTENT FEVER OF QUOTIDIAN TYPE. THE RECORDS INDICATE THE HIGHEST AND LOWEST TEMPERATURES DAILY (Wunderlich.)

1st day.    2d day.    3d day.    4th day.    5th day.    6th day.

In a paroxysm of intermittent fever much may be learned as to the relations of the excretions to temperature, and especially those of the urine, by observing the changes of temperature in very short spaces of time: for example, every fifteen, or even every five minutes (MICHAEL, JONES, RINGER). The rise of temperature is found to begin with, or even to precede, the sensation of chilliness. It takes place at first slowly; and gradually, by about the middle of the period of chilliness, the rise becomes greatly accelerated, lasts through the period of the sensation of great heat, and may even extend into the sweating stage. At the commencement of the sweating stage, small vacillations occur, and continue for a short time; and when the sweating has fairly set in, the decrease of temperature begins, and progresses steadily, without any temporary rise, and with great regularity, decreasing at the rate of 2° Fahr. (or more) every five or fifteen minutes, till it has arrived, after several hours, at the normal heat.

(2.) RANGE OF TEMPERATURE IN A CASE OF INTERMITTENT FEVER OF TERTIAN TYPE. THE RECORDS INDICATE THE HIGHEST AND THE LOWEST TEMPERATURE EACH DAY (Wunderlich).



**Condition of the Urine.**—The observations made on the condition of the urine are divisible into two series, as arranged by Dr. Parkes (*l. c.*, p. 235). (1.) The condition of the urine during the fit, as compared with the urine of a non-febrile period; (2.) The condition of the urine of twenty-four hours during a fever day, as compared with the twenty-four hours' urine of a non-fever day.

During the fit and the apyrectic period the water of the urine is increased in amount during the cold and hot stages: it is most abundant at the termination of the cold or commencement of the hot stage. It decreases during the latter part of the hot stage slowly, and rapidly during the sweating stage. The amount of

increase is variable, and stands in no relation to the quantity of fluid drank, and may be great when this is small (RINGER). The amount of urea excreted by a person with ague, not actually suffering from a fit, is less than in health; but directly the fit commences—that is, at the very first moment of elevation of temperature—or even for some time before this, the urea suddenly increases—an increase which lasts during the cold and hot stages, and then sinks, sometimes gradually, sometimes suddenly, through the sweating stage, or into the commencement of the intermission. It then falls below the healthy average. The amount of increase is very variable, and the type of the fever has no influence upon it; but there seems to be a very close connection between the temperature and the amount of urea. The amount corresponding to a degree of Fahrenheit was greater at a high than a low temperature; and in the fit of each day the same amount was excreted for each degree of temperature (*Med.-Chir. Trans.*, 1859). This increase in the urea must be regarded, in some measure at least, as an indication of increased metamorphosis; and the close relation to the febrile heat certainly implies that it owns only this source, and is not caused by elimination following previous retention (PARKES).

The uric acid is greatly increased during the fit; and after the fit there are often deposits of urates, either spontaneously or on the addition of a drop of acid, and it seems probable that the increase in the excretion of uric acid continues for some time after the paroxysm; and the enlargement of the spleen in connection with this great increase of uric acid is probably not fortuitous (RANKE). The influence of quinine in diminishing the amount of uric acid in health is of interest in connection with its effect upon malaria, and with the condition of the spleen in malarious fever (RANKE, BOSSE). The chloride of sodium is increased during the cold and hot stages to a great degree (TRAUBE, RINGER),—to five times the normal amount; and phosphoric acid is diminished to *one-eighth* (NICHOLSON).

The results are contradictory regarding the urine of a fever day, compared with the urine of a fever-free day. This may be explained to some extent by the relative duration of the fit, compared with the fever-free period; and great differences may arise from the comparative length of the apyretic period on the fever-days; also from the severity or the reverse of the fit, and from the amount of food and drink able to be taken. With respect to abnormal constituents, albumen is found during the fit in a considerable number of cases. Blood in some quantity, and renal cylinders, are seen about as frequently as albumen; and occasionally chronic Bright's disease is a consequence of ague (PARKES, *l. c.*).

**Treatment.**—The treatment of agues varies in a great degree with the complications of the disease; such as with the splenic and hepatic congestions, and the inflammatory affections of these and other organs, which are apt to be established during the existence of an intermittent fever. During each paroxysm, and subsequent to it, the condition of the two important organs referred to ought to be carefully observed; and it ought to be observed, also, whether



of ague. Ramazini relates the case of a patient harassed by an obstinate ague, and who was cured by mercurial frictions administered for syphilis. The influence of splenic disease in keeping up the morbid train of actions of the original fever, and in producing relapses, has been recorded by M. Piorry. In more than 500 cases of ague in which he observed the state of the spleen, he comes to the following conclusions, namely, that the organ is invariably enlarged during the progress of the fever, and that by the use of quinine the spleen diminishes in size; that its reduction in size bears some relation to the quantity of quinine taken; that the effect it produces upon the fever is in proportion to the reduction of the spleen; that the disease is cured simultaneously with the subsidence of the splenic enlargement; and that the fever is apt to recur so long as the spleen exceeds its normal size.

When the fever is severe, accompanied with præcordial oppression, pain, fulness of the spleen or liver, or both, or where there is severe headache, or headache with giddiness, or an oppressive fulness of the chest, a general or a local bloodletting, or both combined, is imperatively demanded, as a means of promoting cure and preventing future evils. The antiperiodic power of bark, quinine, or arsenic, then becomes more easily developed. According to Dr. Copland, such depletion is almost an indispensable preliminary to the administration of quinine or bark, especially in the complicated and congestive forms of the disease. Without such depletion the medicine will either not be retained, or, if retained, it will convert congestions or slight forms of inflammatory irritation into active inflammation or serious structural changes. It is chiefly to a neglect of such a mode of practice that unfavorable consequences have so often followed the use of bark, quinine, or arsenic; for their influence is at first to interrupt secretion, or to over-excite, and subsequently to inflame organs, already loaded, obstructed, and congested. But if blood is to be drawn at all, it should be drawn at the very onset of the hot stage, or that of reaction; and it should be regulated by the constitution, the age, and the habit of the patient, as already explained.

When, on the contrary, the fever assumes a low adynamic form, or when the patient is anæmic, mercurials must be carefully avoided in the treatment under all circumstances, and reliance placed on change of air, quinine, and chalybeates, and improved diet. With regard to liver complication in such cases, the nitro-muriatic acid is to be used internally, in doses of ten drops, three, four, or five times a day, and externally in the form of baths.

With regard to the doses of quinine, some give very large quantities, such as twenty or thirty grains before the expected paroxysm (MAILLOT); others begin to administer the quinine on the subsiding of the paroxysm, and during the sweating stage. According to the experience of Sir Ranald Martin (which has been great in tropical climates), the most rational plan is to give the quinine every three or four hours during the interval of freedom from fever, and in such doses as the urgency of the symptoms may demand. It is to be administered in solution, dissolved by a small quantity of *dilute* sulphuric acid. He also recommends antimony to be conjoined with





by a warm infusion of chamomile, and a mild non-mercurial purgative. Fifteen to twenty grains of the sulphate of quinia should be given in solution, either in a single dose, or in two or three doses at intervals of two or three hours, according to the type and urgency of the case, so that the whole amount shall have been taken two hours before the expected paroxysm. When, from gastric irritability or other causes, quinia cannot be taken by the mouth, it may be administered in an enema, or hypodermically; in the latter case dissolved with tartaric, instead of sulphuric acid, as less locally irritating. Five grains of quinia should be ordered daily, in divided doses, for from fifteen to twenty days, particularly if the disease has lasted any time. It may or may not be combined with iron or arsenic. Where there is intolerance of quinia, arsenious acid, in doses of from one-twentieth to one-thirtieth of a grain, four or five times a day, will be found quite certain. In very young children, arsenic is especially reliable. Numerous substitutes for quinia have been proposed, and have had more or less repute. Amongst these are piperin, tela aranæ (ROBERT JACKSON, CONDIE), beberin (LOGAN, WATT, NICHOLSON), ferrocyanuret of iron (STOKES, FLINT), chloride of sodium (J. C. HUTCHINSON), muriate of ammonia, nitric acid (E. S. BAILEY, W. A. HAMMOND), cornus florida, etc. Of these the cornus florida is the most valuable. Recently the sulphites in full doses, have been used, and with alleged success.

The *congestive* variety of intermittent fever requires prompt and vigorous treatment. To save life minutes must be counted. The chief reliance should be on the immediate administration of large doses of quinia—twenty grains, repeated every half hour, or hour, till there is reaction. If the patient cannot swallow, or there is vomiting, it is to be given in an enema, in larger dose. Chloroform alone, or with sulphuric or chloric ether and camphor and capsicum, is a useful adjuvant. Sinapisms should be applied over the chest, abdomen, and to the extremities, or along the spine; or flannels, steeped in hot water to which mustard has been added, and well wrung; or friction with the hands or a woollen cloth. In the *comatose* form dry cups may be applied to the nucha, and along the spine. But the sheet-anchor is quinia; it is the only antidote to the virulent and quickly-killing poison. After the paroxysm is over, if there are no symptoms of cinchonism—and there usually are not—quinia should be given at intervals, in five- or ten-grain doses, and the patient carefully watched, and kept in bed, until the period of recurrence has fully passed. If about the time of the expected paroxysm the nails should become blue and the finger tips shrivelled, or gaping or drowsiness come on, active means must be at once taken.

*Chronic malarial toxæmia* is best treated by a combination of the chloride of iron, quinine, and arsenic; diuretics; mild saline cathartics, as Seidlitz powders, or the artificial mineral waters—Bitter Kissingen or Pullna; a nourishing mixed diet, including milk, meat, vegetables, and fruit, within the digestive capacity of the stomach; exercise, short of fatigue; and change of climate. Great attention should be paid to the skin; frequent tepid baths should be taken, or the surface sponged daily, followed by gentle and long friction with the hand over the whole body. In some cases the moderate use of *packing*, or the Turkish bath, will be useful. If there is derangement of the chylopoietic viscera, muriate of ammonia alone, or combined with chlorate of potash and colchicum, should be given. Under no circumstances should any preparation of mercury be permitted. Where there is much depression of the nervous system, phosphoric acid, or the hypophosphites, are of great service. In the treatment of malarial toxæmia it is of the first importance to keep the sluices of the system—the kidneys and intestinal canal—open, unless there are contra-



chilliness; or there may be a want of appetite, anxiety, lassitude, pain at the epigastrium, pains in the loins and limbs, headache; slow, small, and irregular pulse; coldness of the skin, and chilliness for one or several days before the commencement of the attack: these are symptoms which usher in a short cold stage. But in other cases the attack is sudden, and the patient, for instance, immediately after a hearty dinner, may be seized most unexpectedly with faintness, vertigo, nausea, confusion of thought; and these almost without a rigor, or a very short one, not exceeding half an hour: a hot stage follows, usually of much greater intensity than that which accompanies the worst forms of intermittent fever.

This hot stage, or period of exacerbation, generally commences in the forenoon of the day, or early in the afternoon, subsiding towards evening, or in the early part of the night, the remissions being generally most complete early in the morning. Sometimes, however, the exacerbations come on towards evening, and last all night, the remissions being then most complete in the forenoon; while, in a few cases, there may be two exacerbations in the twenty-four hours; and these cases are generally the most severe. The exacerbation is usually marked by much cerebral affection, as severe headache, a painfully acute state of every sense, an injected state of the conjunctiva, and great action of the carotid arteries. The pulse, varying from 90 to 120, is generally at first full, but is sometimes from the first small, and generally soft and easily compressible. The tongue is dry, with a white and sometimes yellowish fur, and a bad taste in the mouth. There is generally unquenchable thirst, parched lips, tenderness at the epigastrium, and sometimes pain, with increased dulness on percussion, in the region of the liver. These symptoms are frequently accompanied by delirium, sometimes of a violent character. When giddiness is distressing, and proceeds to delirium at an early period, and runs high, a severe form of fever may be expected. In other cases the patient is oppressed with great drowsiness, lethargy, or coma. The stomach also is often the seat of great pain and uneasiness, followed by vomiting, and the matters vomited are either colorless, bilious, or bloody. The duration of this paroxysm varies considerably, and when the disease is mild it may terminate in six or seven hours; but if severe it may last fifteen, twenty-four, thirty-six, or even forty-eight hours; and Dr. John Hunter once saw a case in which there was no remission for seventy-two hours. Inability to sleep is almost constant. The urine is scanty, high-colored, and of high specific gravity (1024 to 1030), acid, not coagulable by heat (MURCHISON). Albumen was tested for by Dr. Murchison in numerous instances, but never detected; and according to Jones's experience in America, it is very rarely present,—a point of difference, if verified, of great importance as a distinction between severe remittents and specific yellow fever (PARKES). In severe remittent fever Jones found the urea increased, and the uric acid lessened till convalescence, when it again increased, and the pigment was also lessened (PARKES, *l. c.*, p. 242). The fever, however, at length remits, sometimes with sweating, but at other times without any sensible increase of perspiration.



chief indications of the exacerbations are increased restlessness, vomiting, headache, or wandering delirium. In these cases the remissions are not well marked, even from the commencement of the attack (MURCHISON, *l. c.*).

There are great varieties in the degree of severity and type of this fever, more especially as they occur in England, France, Holland, and Germany, compared with those which occur in Spain, Italy, the Mediterranean Islands—or still more so in Africa and the East and West Indies; and accordingly some authors (CRAIGIE) distinguish three varieties,—*e. g.*, (1.) The autumnal remittents of temperate countries, as England, France, Germany, Holland, Hungary. (2.) The summer and autumn remittents of warm countries, as Spain, Italy, Greece, the Mediterranean coasts and islands generally, the Levant, the north of Africa and Asia, and the United States. (3.) The endemic remittents of hot and tropical climates, as in the south of Asia, Central and Western Africa, Equinoctial America, and the West India Islands. Accordingly, remittent fever has received different names from the localities where it prevails. Thus we have the *gall-sickness* of the Netherlands, the Walcheren fever, fever of the Levant (IRVINE), Mediterranean fever (BURNETT), Hungarian sickness, *puka fever* of the East Indies, *jungle fever*, *hill fever* of the East Indies, *bilious remittent* of the West Indies and Mediterranean, Bulam fever, Sierra Leone fever, fever of Fernando Po and Bight of Benin, African fever, and Bengal fever. Prevailing on the borders of inland lakes, as in America, it is sometimes called the *lake fever*. (See page 56 on such nomenclature.)

[Remittent fever is, after intermittent, the most prevalent type of fever in the Middle, Southern, and Western regions of the United States. It is their summer and autumnal epidemic, and unacclimated strangers are very liable to be attacked with it in visiting those sections. From its annual presence and severity in so large a portion of the country, its study is of interest and importance to the American physician. It begins very much like a paroxysm of intermittent fever, with a varying period of lassitude, yawning, and pain in the head, back, and limbs, particularly the calves of the legs; an indescribable uneasiness about the stomach sometimes precedes all the other symptoms; the tongue is coated, and there is a bitter taste in the mouth; the pulse is small, and the action of the heart labored, with increased impulse, and intensity of the sounds. The initial chill may be severe, or moderate, or, what is more frequent, there is a general sensation of cold, lasting from fifteen minutes to a couple of hours, during which there is great thirst, with nausea and vomiting before its termination, particularly if a meal has been recently eaten. The hot stage comes on, with increase of the throbbing headache, which is usually frontal, but occasionally occipital. Violent pain in the back is often complained of. In some cases there is wandering delirium, most frequently associated with a drowsy stupor, shown when the patient is half awake, and passing off when he is quite roused. The pulse is quick, rising in the first paroxysm to 120 or 125, and may be small or full, but rarely hard; the tongue is coated with yellowish fur, and dry, though it may remain moist and almost natural in color; the respiration is hurried and sighing; the thirst is excessive; the urine scanty and muddy. The paroxysm lasts from five to ten hours, when a remission takes place. The first may be decided,



more so than in continued fever; the distinctive type—periodicity in the exacerbations and remissions—is lost; and ataxic and adynamic symptoms supervene. Bronchitis is a common complication.

A favorable change is earlier indicated by the secretions of the mouth and tongue than by any other sign. Even when the tongue is quite dry in the exacerbation, some moisture is apt to appear upon the edges and lower surface during the remissions; and diminished intensity in the coming exacerbations may be inferred from the slightest increase of this moisture during a remission, upon what it was in a previous one.

The *diagnosis* between remittent and typhoid fever is, in general, very easy, even when the intermissions are not well marked, and it has run into the continued type. The absence of many of the chief distinctive traits of typhoid fever will enable us to avoid an error. In remittent fever there is no true eruption, no constant tenderness, with gurgling on pressure, in the right iliac region; the intelligence is nearly always good in the beginning, and may continue so throughout; and the peculiar besotted expression of the face in typhoid is wanting. The access is much more sudden. There is usually an initial chill.

*Malignant congestive or pernicious remittent fever* (*African fever, Country fever, Lake fever*), styled by Dr. Dickson “a hideous and pestilential modification,” prevails in our Southern, Western, and Northwestern States. It begins often as a simple intermittent, and the first paroxysm attracts but little notice. The next chill is more severe; there is extreme coldness of the surface, which is shrivelled, and of a livid hue, and the body is bathed in a clammy sweat, sometimes limited to the face and neck. There is violent gastro-intestinal irritation, with incessant purging and vomiting, the discharges being often mixed with blood, and rarely with bile; the intestinal evacuations have been described as having the appearance of water in which a piece of recently-killed beef has been washed (PARRY). The abdominal tenderness is slight, but a sense of weight and burning heat in the stomach are complained of. The thirst is intense and unquenchable. The respiration is difficult and peculiar—a deep-drawn double inspiration, or double sigh, and one expiration. The pulse is small, thready, and frequent—120 to 140 beats in a minute; but, according to Dr. Boling, the action of the heart continues strong, as shown by the loudness of its sounds and the force of its impulse. There is excessive restlessness, the patient tossing about, and wanting to get out of bed. The intelligence may remain good during the attack, though there is sometimes delirium, and coma may come on after the second paroxysm. Severe headache is very constantly present. If the termination is happy, the restlessness abates, the skin dries, the temperature of the body slowly rises, and the pulse becomes slower and fuller.

A *comatose* variety is met with in the Southern States, which resembles very much the same form of intermittent fever already described. Stupor comes on in the first paroxysm after the cold fit, with dilated pupils and stertorous breathing. As it declines, the stupor passes off, and there is no alarming symptom during the remission. In the exacerbation, the lethargic state returns more marked, and may at once deepen into coma; and this is repeated until recovery or death. The remissions are sometimes very imperfect, and marked only by a temporary and slight abatement in the force, and a diminution of a few beats in the frequency, of the pulse, and cessation of stertor, with yawning and stretching. Dr. Boling relates a case where the patient lay eight days comatose, waking up during the hour of remission on the ninth morning.

The *anatomical character* of remittent fever is a peculiar alteration in





*the fever* must be ascertained,—*i.e.*, whether it be of some hours' or of some days' duration, and whether, when the practitioner sees the patient for the first time, the actually existing paroxysm is at its accession or its decline. It is known by experience that the means of treatment which would be salutary during the first few days cannot be used later to the same effect and in the same amount. There is less tolerance of remedies, and their effects are less therapeutic. Again, it is also known that the means which would arrest fever and save life, if applied at the accession of the paroxysm, would induce a dangerous collapse, or even destroy life, if applied at the stage of its decline, or towards its termination.

The various therapeutic agents which have been employed with various degrees of success in the treatment of remittent fevers are—*emetics, the warm bath, tepid and cold affusions, cold drinks, bloodletting, purgatives, diaphoretics, mercury, quinine or bark, arsenic, wine, and opium.* A review of the prominent modes of treatment of remittent fever, by the most eminent of British army surgeons, has led Sir Ranald Martin to make the following general remark: namely, that a disease so varying in its nature, so general and complicated in its influence on the system, is not to be justly treated by one remedy. Bark and calomel, each a remedy of great power, will nevertheless not succeed in the cure of fever if used exclusively; and so it is with the most powerful of all means, bloodletting. Each remedy must therefore have its proper place in the treatment.

*The first and most immediate object of treatment is to reduce the force and frequency of arterial action during the paroxysm.* If the patient be seen in the forenoon of the first, second, or third paroxysm of an ordinary remittent fever of *sthenic type*, and if he is of a sound constitution, and not beyond middle life, bloodletting from the arm, while the patient is in the recumbent posture, should be practised to the extent of relieving the sufferer from præcordial oppression, from visceral fulness and congestion, or from the intensity of the headache, whichever may predominate. The quantity of blood to be taken is to be regulated by the effects produced, and not by any arbitrary measure in ounces. Evidence of relief from visceral congestion is obtained from the following indications: namely, reduced force and frequency of the pulse, reduction of morbid temperature, and gentle relaxation of the skin. This relaxation of the skin ought not to proceed to sweating, with further symptoms of depression of the vital powers. If it should do so from untoward circumstances, from half a grain to a grain of opium, or from fifteen to twenty drops of laudanum, with as many of chloric ether, should be administered, the object of the administration of either of these medicines being to influence and soothe the heart's action, and to allay gastric or intestinal irritation; and it is only in cases of depression that opium is to be administered thus early in the treatment of the fever.

One general bloodletting will generally be found sufficient to relieve the patient from abdominal or cerebral oppression; and it will further have the effect of simplifying and rendering more efficient.



Dangerous symptoms, such as those just noticed, will sometimes rise suddenly, without any loss of time on the part of the medical attendant, or neglect in treatment. If such symptoms are associated with yellowness of the skin, in persons broken in health, or of feeble constitution, or of dissipated habits of life, or who may have undergone much mental distress, the chances of a fatal termination are imminent.

When the spleen is enlarged, mercury is not to be used in the treatment of the fever; and bloodletting, either general or local, is not borne well. The blood is changed in such cases; it is more or less dissolved, and a general cachexia prevails.

The period of convalescence demands no less careful attention on the part of the medical attendant, especially as to diet and a timely removal from all malarious influences, by a voyage to sea or a change of climate. It is to the mismanagement of convalescence, and a too early discharge from hospital principally, that we must refer the numerous and fatal relapses in the fevers and dysenteries of our seamen and soldiers (MARTIN).

Regarding the method of treatment just described, my friend and colleague, Professor Maclean, writes in the following terms:

“I have been led to take a view of the treatment of malarial fevers generally, and remittent fever in particular, differing from that laid down by many authors. It appears to me that the so-called antiphlogistic treatment, so much insisted on by many writers, is based on the belief that the phenomena observed in a case of remittent fever are consequent on a process of inflammation. It is only on such a belief that antiphlogistic treatment can be justified.

“During the exacerbation of a remittent fever there is violent disturbance both of the vascular and nervous system. Almost every organ, almost every function suffers,—the gastric intestinal membrane is affected, the liver and spleen suffer, the brain is involved, for rending headache and delirium are often present. Is it rational to suppose that an inflammatory process can be going on at one and the same time in all these various organs? Do the appearances observed post-mortem give any support to such a doctrine? If not, on what principle can spoliative treatment be justified? Is it not rather the case that this terrible disturbance of so many organs is due to the presence in the blood of a subtle poison acting on them all? If so, surely the guiding principle of the physician in his treatment should be to counteract this poison, to neutralize it, or to expel it from the system, and so to prevent a recurrence of the exacerbation. This is the principle on which I have long acted, and I am satisfied that it is at once a safe and successful one. In quinine we have such an antidote—a therapeutic agent of unrivalled efficacy, which, if skilfully used, will rarely disappoint the expectations of the practitioner.

“It is always, of course, advisable to have the bowels thoroughly evacuated; and if the patient is seen when his stomach is loaded, it is well to evacuate its contents by an emetic. In ardent remittents, however, there is generally little call for this, as obstinate vomiting is almost always a troublesome symptom. This done, the period of remission must be watched for, and, the moment it arrives, quinine in a *full dose* should be given—not less than fifteen grains in the case of an adult. If



the system of treating this fever by saturating the system with mercury I enter my strenuous protest. I know nothing more deplorable than the condition of a patient whose constitution, already depressed by the presence of malaria, is further saturated by another poison which acts as a powerful ally of the first."

In the asthenic form of remittent fever, such as that so well described by Dr. Murchison as prevailing in Burmah, it is necessary to exercise great caution in depletion. All the cases he relates which had been freely bled exhibited the most aggravated typhoid symptoms, and most of them died. Even in the instance of young and robust recruits, low adynamic typhoid symptoms were sure to supervene in a short time after bloodletting; and, even although it gave temporary relief, it was certain to aggravate, if not to induce, the subsequent typhoid condition. If the headache is very severe and the pulse full, a few leeches may be applied to the temples at the commencement of the attack; but if the hair be cut short, or shaved off the scalp, cold lotions applied to the head, or the cold douche kept up for ten minutes at a time, gives great relief, and is the preferable remedy (MURCHISON). As soon as possible after the commencement of the paroxysm the bowels should be cleaned out with a purgative of calomel and compound jalap powder; or by colocynth, antimonial powder, and calomel. If typhoid symptoms betray themselves, stimulants, such as wine and brandy, must be given; but, as in intermittent fever, "quinine is undoubtedly the sheet-anchor," and it is best given, as in the former fever, in one large dose of twenty grains at the very commencement of a remission. Carbo-azotic or picric acid has been lately introduced as an active remedy in the treatment of malarious fever. Prepared by Calvert, of Manchester, it is of a light yellow color; and in doses of two grains, cautiously repeated, it is to be pushed till the patient gets yellow-skinned.

#### MALARIOUS YELLOW FEVER—SYN., FEBRIS ICTERODES REMITTENS.

**Definition.**—*Febrile phenomena due to malaria, in which the exacerbation and remission are so connected that the fever resembles a continued fever, and is characterized by great intensity of headache and yellowness of the skin (COPLAND, DICKENSON, BOOTT); but in which the urine is not suppressed, and continues free from blood or albumen.*

**Pathology.**—It immediately results from the history of yellow fever, that in its malarious form it is the product of the coasts of the West India Islands, the American equinoctial continents, several districts in Spain, and the west coast of Africa. All over the Caribbean Sea the disease takes place sporadically, or in insulated cases every season, more or less numerous according to the subjects and the number of new visitors, and there never is a season in which a few cases do not occur. At Vera Cruz, Havana, and other towns on the Spanish Main, malarious yellow fever invariably attacks Europeans or Canadians who may land there between the months



The following observation was made by Sir John Pringle on the fevers of Walcheren and South Beveland in 1747:

“These epidemic fevers, by reason of the great heats of the season, not only began more early than usual, but were fully as fatal to the natives as to us. But Commodore Mitchell’s squadron, which lay all this time at anchor in the channel between South Beveland and Walcheren, in both of which places the distempers raged, was neither afflicted with fever nor flux, but amid all that sickness enjoyed perfect health—a proof,” he says, “that the moist and putrid air of the marshes was dissipated or corrected before it could reach them” (*Diseases of the Army*, p. 58).

The very same observation was made at the very same spot, fifty-two years afterward, by Sir Gilbert Blane:

“I had, in the course of this service (at Walcheren, in 1809), an opportunity of observing the extent to which the noxious exhalations extended, which was found to be less than I believe is generally known. Not only the crews of the ships in the Road of Flushing were entirely free from this endemic, but also the guard ships stationed in the narrow channel between this island and South Beveland. The width of this channel is about six thousand feet; and although some of the ships lay much nearer to the one shore than the other, there was no instance of any of their officers or crew being taken ill with the same disorder as that with which the troops on shore were affected” (*Med.-Chir. Trans.*, vol. iii, p. 27).

It is now also generally believed that the malarious form of yellow fever cannot exist except in places where the average range of temperature is high throughout a considerable part of the year; and for this reason it is believed that it will not become a disease of this country. Sir Gilbert Blane asserted that it never appeared either in tropical climates or in the temperate latitudes, unless when the atmospheric heat has been for some time steadily at or above 80° Fahr., 21° of Reaumur, or 26.67° Cent.; according to Humboldt, 75° of Fahr., or 24° Cent.; and according to Matthei, 72° Fahr., or more. The disease is also found not to prevail in mountainous situations. According to Humboldt, it has never ascended to 3044 feet above the level of the sea, and according to Sir Ranald Martin, never above 2500 feet; and below the former limit the Mexican oaks do not flourish, showing that the constant average temperature below this is of a tropical character. In Jamaica, according to Dr. Craigie, it rarely ascends 1600 feet above the level of the sea (Dr. Lawson’s instance of the outbreak at Newcastle being considered an instance of “specific yellow fever”). “In Jamaica the medium temperature of Spanish Town in the hottest months is about 85° Fahr., or between 83° and 85°; and in Kingston it is much the same, ranging from 85° to 90°, and rarely falling below 80° from May to the end of September. At the more elevated parts, however, the temperature diminishes, being only about 70° at Stony Hill, elevated about 1300 feet; at Cold Spring, 4200 feet above the level of the sea, only 60°; and at the summit of the Blue Mountains, which are estimated to be 7200 feet above the level of the sea, the thermometer is found





[TYPHO-MALARIAL FEVER.—*Chickahominy Fever, American Camp Fever.*  
(DR. CLYMER.)

**Definition.**—*An idiopathic fever of mixed type, caused by a combination of paludal and pythogenetic influences, with marked remissions and exacerbations at the beginning, and, after a variable period, becoming continuous; attended with early prostration, diarrhœa, and subsequently extreme adynamia: the characteristic lesion is enlargement and ulceration of the solitary intestinal glands.*

**History.**—This form of fever attracted attention first in 1862, as the Chickahominy fever, from its prevalence in the Army of the Potomac at that time, but has since been common whenever our armies operated in malarious regions, amongst men saturated with paludal poison, exhausted by over-exertion and insufficient rest, imperfectly nourished, exposed to the action of animal effluvia from the decaying bodies of both men and brutes, and drinking water impregnated with the products of common putrefaction. These coincident causes, tending to lower the vital forces and corrupt the blood, produce a compound disorder, in which the combined action of paludal, pythogenetic, and scorbutic influences are evident, and which varies in type, as one or other of the determining conditions is predominant. The name *typho-malarial* was proposed and first used by Dr. J. J. Woodward, U. S. A. (*Outlines of the Chief Camp Diseases of the United States Armies*: 1863).

**Symptoms.**—The attack is generally sudden, beginning with a chill; there are headache, anorexia, thirst, diarrhœa, and sometimes epistaxis. The tongue soon becomes coated with a thick, dry, brown fur. For some days there are distinct remissions and exacerbations; in the early part of the second week they become less marked, though they may persist throughout the attack. Regular remissions very commonly again take place on the approach of convalescence. Diarrhœa is apt to be troublesome and persistent, as the continued type is developed; the mouth is coated with sordes; an herpetic eruption may appear about the lips and nose; wakefulness is constant, with low muttering delirium; tympany, rare in the first week or ten days, occurs; and purpuric blotches, or petechial spots, with hemorrhage from the bowels, gums, mouth, and nostrils; and all the phenomena of a low form of fever now set in. Matter resembling coffee-grounds is sometimes vomited towards the last. If the disease is to terminate favorably, regular remissions again happen, generally in the forenoon, with evening exacerbations. Congestive pneumonia, bronchitis, and parotitis are the intercurrent affections, particularly bronchitis. An attack lasts from three to five weeks; and convalescence is very lengthened.

**Anatomical Characters.**—The characteristic lesion is enlargement of the solitary follicles of the small intestines. There may be universal congestion of the mucous membrane of the small intestines, more marked in their lower part, or there may be only congestive patches of variable size in the ileum, the solitary follicles being enlarged from the size of a pin's head to that of a pea, and black with pigment deposit; they sometimes look like yellow mustard-seed sprinkled on a red ground; their apexes are sometimes ulcerated. The mucous membrane of the colon may be of a slate color, with patches of congestion and spots of ecchymosis, or it may be streaked of ash and dark red. Small ulcers are occasionally found in the follicles of the colon, cæcum, and appendix vermiformis. The patches of Peyer are generally unaltered, though they may be con-



endemic in particular districts, but that it occasionally spread over large portions of country, while still later, in the year 1557, it was found to prevail epidemically, not only over the whole of Europe, but even over the whole of the northern hemisphere, beginning in Asia and proceeding westward till it terminated in America. In the eighteenth century, having advanced westward till it reached the Elbe, it passed over the intermediate countries and reached England, where the stream broke into two branches, the one crossing the Atlantic to America, while the other retrograded southeast through France, Spain, and Italy, till it was lost in the Mediterranean—a course similar to that described by cholera.

Influenza has occasionally originated as far eastward as India, but more commonly it has broken out in the north of Europe, as Moscow, Warsaw, or Dresden. It seems probable that, like the poison of Cholera Indica, its spread may be limited to a small number of primary *foci*; for we find in every volume of the *Calcutta Transactions* accounts of some catarrhal fever spreading for a season along the banks of some principal river, and then subsiding; so that it is evidently only occasionally and at long intervals *erratic*, as in 1729, 1743, 1775, 1782, 1831, 1833, and 1837. The influenza, therefore, is both endemic and epidemic; and, in the latter case, we find it, at least in Europe, spreading from east to west, prevailing in the depths of winter as well as the heights of summer, lasting nearly the same space of time in the different towns and cities it attacks, or from four to six weeks, affecting contiguous places in different degrees and at different times.

On looking to the habits of this poison, it is probable that its actions are not limited to man; for in most years, when influenza has been epidemic, a similar disease has been epizootic, especially among horses and dogs, as in the years 1728, 1732, and 1775. It is a disease of extraordinary rapidity of progress; and as its diffusibility is great, so are its periods of recurrence frequent—those cycles of its visitation which are as yet beyond our comprehension to explain.

**Pathology.**—A specific poison is believed to be absorbed, and to infect the blood, when, after a period of incubation varying from one to two or three days, or even to two or three weeks, it produces disordered functions of the great nervous centres, causing great general depression, extreme debility, together with slight or severe remittent fever. The specific actions of this poison are on the mucous membrane of the eyes, of the nose, and of the bronchi; in a smaller number of cases on the mucous membrane of the fauces, causing sore throat; and in a still smaller ratio on the substance of the lungs and on the pleura, causing inflammation of those organs. In most instances the disorder terminates in diarrhœa. These different pathological phenomena vary in frequency and complexity in different seasons and places.

In most cases, where the poison is of sufficient intensity to produce fever, the type is remittent in this country, with exacerbations in the evening. Its usual duration is two, three, or four days, when it terminates in an abundant sweat, and which not unfrequently



ably described by Dr. Peacock) was from three to five days in the mild forms, and from seven to ten in the more severe.

In mild cases such phenomena constituted the whole disease, and the patients recovered about the eighth or tenth day, after suffering for a few hours from sharp diarrhoea or profuse perspiration. In many instances, however, the patient, in addition, suffered from mild or severe sore throat; or cough came on, and continued for many weeks. In a few cases the symptoms were of a more aggravated character, the fever being more marked, the pulse accelerated, the skin hotter, and the cough more troublesome; and these conditions have often been followed by inflammation of the lungs.

The pulmonary complications may be arranged into four forms,—(1.) Capillary bronchitis; (2.) Bronchitis supervening on tuberculous disease of the lungs; (3.) Bronchitis with disease of the heart or aorta; (4.) Pneumonia.

The accession of capillary bronchitis is indicated by the chest symptoms becoming more severe and the cough paroxysmal, and the dyspnoea at first quite disproportionate to the cough and to the physical signs. The expectoration is scanty, and consists of small yellowish pellets, forming tenacious masses of a peculiarly nodule form. The pulse becomes rapid (120—140), the tongue covered with a white-brown fur, and prostration is extreme. The only auscultatory signs are roughness of the inspiratory murmur, with occasional sibilus, and slight crepitation at the back. There is soreness and contraction of the chest, but no acute pain. Crepitation, unattended by dulness on percussion, soon extends over a greater or less extent of both lungs; and the dyspnoea speedily becomes so intense as to prevent the patient from lying down, the lividity of the lips and face increases, and the eyes become prominent. The cough is now very frequent, the sputa very viscid, of a greenish-yellow color, without air-bells, and often streaked with blood. The respirations are quickened; but there does not appear to be any uniform connection between the extent of the disease and the disturbed ratio of the pulse and respiration movements. The general rule is, that the respirations are relatively more quickened than the pulse (PEACOCK, PARKES). The physical signs soon become modified by rapidly developed emphysema of the lungs. Generally, it may be said that the *capillary bronchitis* of *influenza* is distinguished from *pneumonia* by the greater severity of the general symptoms; by the tendency of the fine crepitation of the early stage to pass into subcrepitant and mucous râles, rather than to give place to evidences of condensation, and by the peculiar characters of the cough, which is paroxysmal, and not attended by pain; and, lastly, by the character of the expectoration, which consists of whitish viscid pellets, cohering into irregular masses, and destitute of the glairy adhesive character, russet color, and small air-bubbles of pneumonia expectoration (PEACOCK).

Inflammation of the substance of the lungs seldom occurs till the second or third day, and more commonly not till the fifth or sixth; and, although generally, is not always preceded by shivering, or even bronchitis. The pneumonia in some years has been charac-



the higher and more healthy districts. The epidemic of 1847 was much more fatal in the insalubrious parts of London than in those less unhealthy; and according to Dr. Peacock's experience, the mortality of influenza was owing more to the condition in which the disease found the patient, than to any inherent power of the poison itself—a result conformable to general experience and the returns of the Registrar-General (PARKES).

The nature of the "*epidemic influence*" which gives rise to *influenza* is quite unknown. Sudden changes of temperature appear to assist the development of the influenza poison; and exposure to cold predisposes the individual to the disease—which seems to be a disease especially of the higher latitudes.

**Susceptibility Exhausted.**—Few persons suffer more than one attack of influenza in the same epidemic, although many relapse; and one attack of this disease in no degree protects the constitution from a second attack in another epidemic.

**Prognosis.**—Children and persons under forty die in a very small proportion, unless in a previous state of ill-health. The mortality, however, among the aged has in every country been great from this disease. It has been remarked, also, that the disease, if not fatal in itself, left the patient, of whatever age, often greatly debilitated in body and depressed in spirits, and that those with tender lungs who suffered from it frequently fell into phthisis, or continued to cough for several months afterwards, so that a complete recovery was often long and tedious.

**Treatment.**—As a general rule, the great majority of cases in epidemics of influenza have scarcely required any medical treatment. In that of 1782 it was observed that "many, indeed, were so slightly indisposed as to require little or no medicine; nothing more was wanted to their cure than to abstain for two or three days from animal food and fermented liquors, and to use some soft, diluted, tepid drink. A lenient purgative at the beginning of the disease was useful in moderating the fever, and nature seemed to point out the repetition of it afterwards when there was pain in the stomach and bowels, and a tendency to diarrhœa. The same was observed in 1762. Nothing, likewise, was observed so successfully to mitigate the cough as a gentle purge to open the bowels, and afterwards to give a gentle opiate at night. In the year 1837 it was also remarked that, as long as the symptoms were limited to cough, hoarseness, headache, or other pains moderate in degree, the patients all recovered by putting them on a low diet, by attending to their bowels, and confining them for a few days to the house; and, if more was attempted it was quickly found that the disease ran a course scarcely influenced by medicine. A smaller number, however, required medical attendance, either from the severity of the bronchitis, the occurrence of pneumonia, of angina or of severe dyspnoea, of the disordered state of the bowels, or more frequently from the debility induced by the disorder.

Bloodletting is always hurtful. It does not relieve the fever, and increases the nervous depression. In general, when pleurisy, bronchitis, or pneumonia may supervene, leeches to the chest, or





## WHOOPING-COUGH.

LATIN, *Pertussis*; FRENCH, *Coqueluche*; GERMAN, *Keuchhusten*—Syn., *Stickhusten*; ITALIAN, *Tosse convulsa*—Syn., *T. canina*.

**Definition.**—*An infectious and (sometimes epidemic) specific disease, preceded and accompanied by fever of variable intensity; attended in the first instance by catarrh, and subsequently by paroxysmal fits of coughing, which occur in numerous short, rapid, spasmodic, convulsive movements of expiration, suddenly followed by a prolonged inspiration, marked by a characteristic sound of a sonorous kind, and variously named the “kink,” “hoop,” or “whoop.” These paroxysms of expiratory and respiratory convulsive movements alternately recur several times, till the fit ends by a quantity of mucus being brought up from the lungs, or till the contents of the stomach are evacuated.*

**Pathology and Morbid Anatomy.**—The theory of this disease is that a specific morbid poison produces slight primary fever, which for the most part subsides on specific or secondary actions being established. These are catarrh, followed by a peculiar cough and vomiting, ascribed to irritation of the *vagus nerve* by the specific poison. The disease is a “specific pulmonary catarrh;” but very different opinions have at various periods been entertained as to its nature. Its origin appears to have been comparatively of no very distant date, Sprengel not having been able to trace it beyond 1510, when it was endemic in Paris; but its epidemic character was not determined till 1580.

Like other diseases of this class, it appears, as a rule, but once during life, and attacks chiefly infants and children. Dr. Watson gives an instance of a child born with whooping-cough. There are instances, however, of its occurring not only late in life, but also a second time (HEBERDEN). Blache gives a remarkable instance of a grandfather and grandmother catching whooping-cough a second time from their grandchild, all of them laboring under the disease together. Some consider the disease to be a specific affection of the nervous system; others, that it is a catarrh; but both these pathological conditions coexist in whooping-cough. Inflammation does not necessarily accompany the disease, although a state of the mucous membrane exists by which it is morbidly irritable, or susceptible to impressions.

Pathologists have also ascribed the complaint to a morbid condition of the pneumogastric nerve—an explanation supposed by some to be confirmed by the circumstance that that pair of nerves is sometimes found red, with the medullary matter altered in color, dense in texture, and of cartilaginous firmness (KILIAN, AUTENRIETH). Others believe that a specific poison acts on some part of this nerve (TODD).

The results of nineteen post-mortem observations made by Dr. Graily Hewitt during a recent epidemic of this disease (1855), in children varying from one month old to four years, showed the chief lesion to be collapse of the lung-substance—a condition also



trachea is the indication of a convulsive cough coming on; and no doubt this is the warning which young children recognize and dread as the harbinger of a paroxysm, which suggests to them the necessity of seizing something for support during the fit of coughing, which almost immediately commences. The irritation is attempted to be got rid of by coughing; and in the expiratory efforts the air is expelled with great violence, and so repeatedly and irresistibly that the lungs are ultimately almost emptied of air. At the conclusion of these expiratory efforts the condition of the lungs resembles that produced by *asphyxia*. A sudden inspiration now necessarily and suddenly follows, the air being drawn through the glottis by the gasping patient, with a force and velocity which gives rise to a shrill, sonorous sound, not unlike the crowing of a cock, and which has been variously named a *kink*, a *hoop*, or *whoop*; and the disease has accordingly received various names, such as *kink-host*, *hooping-cough*, *whooping-cough*, *chincough*. The anxious and distressing inspirations are scarcely completed when the convulsive expirations of the cough are again renewed, and again followed by the gasping and crowing inspirations, till a quantity of mucus is brought up from the lungs, or till the contents of the stomach are rejected by vomiting. Such are the phenomena of the fit or paroxysm of whooping-cough. After it is over, the patient in ordinary cases appears to be but little affected, and returns immediately to play, or to any other occupation which takes the attention at the time.

When these phenomena are prolonged, secondary effects are produced, whose morbid appearances have been noticed. The immediate consequence of the violent fits of coughing is to interrupt the free transmission of blood through the lungs, and the return of blood from the vessels of the head. This causes not only the turgidity, swellings, redness, and lividity of the face and eyelids which attend the fits, but also the discharges even of blood from the mucous surfaces of the nose, ears, or eyes. The little sufferer may shed tears of blood.

Whooping-cough varies greatly in intensity, and most authors divide the group of symptoms into three stages. The first stage comprehends the period from the first symptoms of illness until the "*whoop*" confirms the convulsive nature of the cough. This is the period of development or evolution. The second stage commences as soon as the nature of the cough is determined, and lasts till the violence of the cough and the danger of secondary complications is past. This is the period of spasmodic paroxysms characteristic of the disease. The third stage comprehends the convalescence of the patient, until the final and happy termination of the disease; or the occurrence of any event which may destroy the sufferer.

When the convulsive stage of the disease is fully formed, and the series of fits or paroxysms of severe coughing occur at uncertain periods, during the interval the patient generally enjoys his usual health, recovers all his gayety, returns to his play, and relishes his food with a good appetite.

A paroxysm or fit of whooping-cough comprehends the following phenomena:



is to a certain degree convalescent. The duration of this second stage is from two to six or eight weeks.

The third stage commences with the convalescence of the patient, when the paroxysms become milder, the intervals longer, the expectoration thicker and more opaque, greenish, or pus-like, and more like ordinary catarrh. The vomiting ceases, and the general health of the patient begins to improve greatly. The duration of this stage however, is often long and variable, and the cough may still harass the patient for many weeks, or even many months. It is to this stage that the term chronic is usually applied.

The disease lasts from six to eight weeks; but there are exceptional cases, which on the one hand get well in a week, and on the other hand may continue several months, or even a year. "The general duration of the disease is directly proportionate to the duration of the prodromata;" and the more quickly the convulsive cough makes its appearance, the more quickly does the disease subside (TROUSSEAU).

Many accidents may arise to complicate the symptoms of whooping-cough, and to increase the danger, as inflammation of some of the tissues of the lungs, of the mucous membrane, of the stomach or intestines, or of the serous membranes of the brain.

Inflammation of the minute bronchia is the most usual complication of this disease—*capillary bronchitis*, or *peripneumonic catarrh*. The form of inflammation may be that in which the secretions are in defect, so that the mucus is not only greatly diminished in quantity, but is thick and viscid, teasing the patient with fruitless efforts to free it from the air-tubes, and thus causing a frequent recurrence of the paroxysm. In other cases it may assume the form of purulent inflammation, the pus secreted being formed into sputa, and moderate in quantity; or it may be thrown up pure, as from an abscess, and so enormous in quantity as to amount to one or two pints in the twenty-four hours. The inflammation of the bronchial membrane may spread to the substance of the lungs, when the danger, as well as the symptoms, of some of the various forms of pneumonia will be added to the disease; but the most formidable accident is when the pleura is inflamed, for then the patient's sufferings during the paroxysm are fearfully increased, from the agonizing pain inflicted during the paroxysm of the cough. These lesions are the most frequent causes of death in cases of whooping-cough.

The mucous membrane of the stomach and intestines is often the seat of inflammation; and this is denoted by pain in the epigastrium, and by the suppression of the glairy fluid thrown up by vomiting, so that on the termination of the fit the patient often lies in a state of complete exhaustion, unable to discharge anything either from the stomach or lungs, or even to "*whoop*;" and he is then said, in popular language, to labor under the "*dumb-kink*."

In mild cases the bowels are little affected in this disease, except that the patient sometimes passes his fæces during the paroxysm. In severe forms the stools are often either black and offensive, or they consist of a colorless mucus, the latter evidently depending on an inflamed state of the mucous follicles.



The poison of this disease may coexist with many other poisons, and in this case they often greatly influence each other's actions. *Small-pox* and *whooping-cough* have often coexisted; and a very common and fatal combination is *measles* and *whooping-cough*. *Whooping-cough* and *cow-pox* are not unfrequently combined. Indeed, the lower classes erroneously look upon vaccination as in many instances a cure for *whooping-cough*.

**Period of Latency.**—The disease has a stage or period of incubation, but our knowledge of the extent of this period is at present extremely imperfect. The disease never shows itself immediately after exposure to contagion, but a certain number of days (five or six) elapses before the symptoms of catarrh are to be observed.

**Prognosis.**—The proportionate number of deaths to recoveries in *whooping-cough* is not determined, but greatly varies in different years; for in one year hardly a death will occur from the disease in a large city, while in another year many children will die. In general, however, the milder forms of the disease are rarely fatal, while the more severe and protracted cases very commonly are so. Lombard thinks station in society greatly affects the mortality; for he says that of ten fatal cases nine belong to the poorer classes. The reports of the Registrar-General show that the mortality is greater from this disease in towns than in the country, being in the metropolis, in 1838, .111 per cent., while in England and Wales it was .061. In the year 1839, also, it was for the metropolis .061 per cent., while for England and Wales it was .053. Lombard gives the ages of forty fatal cases as follows:

AGES.	CASES.	AGES.	CASES.
From birth to 6 months, . . .	6	From 4 to 5 years, . . . . .	2
" 6 to 12 months, . . .	7	" 5 to 6 years, . . . . .	2
" 1 to 2 years, . . . . .	10	Above 6 years, . . . . .	0
" 2 to 3 years, . . . . .	6		—
" 3 to 4 years, . . . . .	7	Total, . . . . .	40

Danger from bronchial inflammation is to be dreaded rather towards the end than the beginning of the disease. Convulsions are apt to occur if dentition is going on at the time; and if they arise from the congestion or effusion within the cranium, the case is generally fatal. The number of paroxysms which a child may have in twenty-four hours is the best basis of prognosis. Twenty fits in twenty-four hours denote a very mild case; when more violent, forty to fifty paroxysms may occur in that period; and when the number of paroxysms exceeds forty, the case is a serious one, and prognosis grave.

If a predisposition to tubercle exists, *whooping-cough* may determine the development of *phthisis*.

**Treatment.**—On the invasion of the disease, beyond putting the patient on a low or very moderate diet, and attending to the daily action of the bowels, there is little occasion for medicine.

The "*whoop*" having confirmed the nature of the affection, and the second stage being established, the disease will run its course, and one of two indications of treatment may be followed. The





rule in all cases, to be combined with it. The selection of the particular medicine is perhaps unimportant, and any vegetable or saline purgative will perhaps answer equally well, as the *confectio sennæ*, *rhubarb*, *castor oil*, or *manna*. The neutral salts, however, sit easiest on the stomach, and (as the medicine must be continued) are the most agreeable to the patient. *Opium* is a dangerous remedy, and is liable to the objection of being apt to check the mucous secretion.

If at the outset or afterwards the cough is very suffocative, an emetic is useful. Five to nine grains of sulphate of copper dissolved in three ounces of distilled water, and a dessert-spoonful every ten minutes is the most efficient (TROUSSEAU).

Nitric acid, in the following formula, has been found of service:

Acid. Nitrici diluti, fʒxij; Tinct. Cardam. comp., fʒiij; Syrup, fʒiiiss.; Aquæ, fʒj; *misce*. Of this mixture, one or two small teaspoonfuls may be given every two hours (GIBB).

Towards the close of the second stage the symptoms may become unfavorable, and cerebral irritation, with convulsions, or inflammation of the membranes of the brain, of its substance, or of the tissues of the lung, or of the alimentary canal, may complicate the disease, and then the treatment of the case is always exceedingly difficult, and frequently unsuccessful.

If convulsions should come on suddenly, and without headache or other symptom of inflammatory action, small doses of any opiate, and *mustard poultices* to the feet, may relieve the patient; but should convulsions still continue, an *asafætida injection* may be administered. It often happens that convulsions are combined with a suppression of the vomiting, and of the usual glairy discharge; and in these cases *leeches*, followed by a large *linseed poultice*, should be applied to the epigastrium. If the unfavorable symptoms should advance, and headache or other symptom show an affection of the membranes of the brain, *leeches* should be applied to the temples and *cold* to the head.

When the poison excites inflammation of the tissues or substance of the lungs, *bleeding* to a limited amount by leeches may be required; but we should be satisfied with such mitigation of the symptoms as may obviate immediate danger, and even that is not always obtained, since the affection is not to be subdued by bleeding, as in simple inflammation; for, being dependent on the action of a morbid poison, it will run a definite course. Blache bled in nine cases, either with the lancet, by leeches, or by cupping, and in one case no less than five times; yet, he adds, with a desolating want of success, and eight out of the nine cases terminated fatally. This result makes him add an axiom, in which every practitioner will agree, that there is in severe *whooping-cough*, as in *typhus*, *cholera*, and many other affections, an unknown element which modifies and gives a specific character to all these intercurrent inflammations.

If the intestinal canal be affected, some purgative, combined perhaps with *calomel*, may be necessary to act on the bowels and free them from their contents; and if the stools be white and muciform, and the patient not relieved, an enlarged state of the follicles may



tients when removed from large towns to their environs, for even in a few hours they have been known to recover from an apparently hopeless state. A sail across a river is also beneficial, although the distance may be short.

**Dietetic and General Treatment.**—The patient should not be allowed animal food from the commencement almost to the termination of the disease *in its acute form*. It is desirable also that the temperature of his apartment should be regulated, and that he should not be exposed to any considerable or sudden change from heat to cold. In mild weather also, if no local symptom forbids, he should be permitted to take exercise in the open air. He should likewise wear flannel.

There are no known means of prevention, except an entire removal from every source of contagion.

### MUMPS.

LATIN, *Parotides*; FRENCH, *Oreillon*; GERMAN, *Ziegenpeter*—Syn., *Mumps*; ITALIAN, *Parotitide*.

**Definition.**—*An inflammation of the parotid and salivary glands, probably specific, and certainly in some cases contagious and epidemic.*

**Pathology.**—It is most common in male children; and is less frequent after puberty; and second attacks of the disease are rare. The disease is sometimes epidemic, and in certain localities it prevails rather than in others; so that local causes may have to do with its propagation and maintenance.

The disease often occurs also in the course of severe fevers (typhus and enteric); and it has been noticed in cases of cholera. It is a result also of ptyalism from iodine or from mercury. Dr. John Harley gives the following example of its direct propagation from person to person. “A medical student had *mumps* in London, at a time when his mother was staying with him. They remained in town till the swelling disappeared, and then went—a hundred miles into the country—home. There was no *mumps* in that neighborhood; but a fortnight after their arrival one of the children was taken with the disease, and it afterwards successively affected, at regular intervals of a fortnight, each member of a large family” (Hooper’s *Physician’s Vade Mecum*, 7th edition, p. 558).

**Symptoms.**—Febrile phenomena, associated with pain and uneasiness in the region of the parotid. The pain on moving the jaw soon becomes so great that mastication becomes impossible in severe cases. Considerable fulness and soreness prevail at the angle of the jaw over the malar region and region of the parotid, on both sides generally. Beneath one or both ears redness prevails, with pain on pressure; and the pain becomes so great as to prevent sleep at night in severe cases; and in such cases the swelling generally extends to the submaxillary glands, and to the tonsils, and neighboring parts of the pharynx, so that swallowing becomes very difficult and painful. The region of the swollen glands becomes tense and glossy; sometimes of dusky livid hue; and when both sides



siderable extent; and since that time many excellent monographs have been written on the subject in our own country, among which those of Hunter Semple, Chatto, Wade, Ernest Hart, Greenhow, Sanderson, and Jenner are conspicuous.

**Pathology and Morbid Anatomy.**—In this disease, as in many others of the *miasmatic* kind, the *general* or the *local* symptoms may predominate, giving special features to each case; and the patient may die from the severity of the general disease, or from the severity of some one of the local lesions.

The mucous membrane covering a tonsil may be the primary seat of the characteristic local exudation, or the arches of the palate, or the posterior surface of the soft palate, the uvula, the nares, or the pharynx may be the primary seat. At first there is redness and swelling; and the normal mucous secretion is so altered in its physical properties that it adheres by its own increased viscosity to the mucous membrane. A white or gray patch now forms on the membrane, which indicates the presence of a layer of lymph on the reddened surface.

The layer of lymph may thus spread from one or from several centres over the reddened surface; and this redness may involve the whole mucous membrane within reach of the eye. The lymph which grows upon this reddened surface may descend into the larynx, the trachea, and the bronchi. Dr. Stokes has recorded a fatal case, in which the tongue, tonsils, pharynx, epiglottis, larynx, trachea, and right bronchus were more or less thickly coated with the deposit, even as far as the fourth or fifth bronchial ramification, while the left bronchus remained quite free from it. The right lung was œdematous and consolidated, the left comparatively healthy (*Dub. Jour. Med.*, Feb., 1863). Dr. Jenner has known the diphtheritic exudation to extend into the œsophagus and stomach (*Diphtheria; its Symptoms and Treatment*, by Dr. Jenner, p. 4). If the lymph be torn from the mucous membrane, a raw, bleeding surface is exposed, which in a few hours is again covered by a new layer of lymph. The lymph of diphtheria has a variety of appearances. Sometimes it is granular, with very little consistence or tenacity. Sometimes the part is covered with a pulpy substance of a white or gray color; but this pellicle is constant in some form or other, and is possessed of the power of reproducing itself. It is this specific exudation which establishes the disease as one *sui generis*, and to which Bretonneau gave the name of "*Diphtheritis*," and which has been subsequently modified to "*Diphtheria*." The latter term has the advantage of being the shorter word, and is that adopted by the Registrar-General. Etymologically, the terms are derived from *διφθέρα* *vel* *διφθερίς*, signifying the prepared skin of an animal; while *διφθερίτης* *vel* *διφθερίης* signifies that which is covered with a fur, or with a leathern coat.

In microscopical characters it does not appear that this "fur," "pellicle," or "false membrane" of diphtheria can be distinguished from the concrete exudation on blistered surfaces, or that which forms in the angina of scarlatina (EMPIS). The commencement of the formation of the pellicle is in reality an act of coagulation. The



the renal complication, as affording an anatomical explanation of the fact, that, in many cases of diphtheria in which death occurs neither by suffocation nor by septic poisoning, it cannot be due to local lesion. Bonchut considers it a sign of the commencement of purulent infection in diphtheria, and coincides with very great gravity of the disease. The blood then assumes the tinge of bistre; and numerous masses of pulmonary apoplexy may be found after death, resembling those which precede the development of metastatic abscesses in the lungs. Mr. Wade says (*The Lancet*, 1862):

“When my attention was first turned to diphtheria, the doctrine of Bretonneau was paramount, that diphtheria is a local disease, and for the most part always remains so, infecting the system, if at all, only by absorption of putrescent matters from the throat, and, consequently, all treatment was to be local. I was struck early by the fact that many slight cases die; and I resolved to make a full dissection as soon as opportunity should offer, without any preconception as to what I should find. In the first case I found (after about seven days' illness) a pair of white kidneys, such as one finds after scarlatinal dropsy. The spleen was occupied by a similar deposit. After this I turned my attention to the kidneys during life, and found not infrequently *albuminuria*. I have never said (because I never believed) that *albuminuria* and *uræmia* were convertible terms; but I do say that I have seen many cases of diphtheria which presented distinct symptoms of *uræmia*—comatose or comatoid phenomena—coincidentally with an obvious diminution of the urinary secretion, and relieved (and that very suddenly) by restoration of the secretion. I looked upon the discovery of renal complication as important, for two reasons,—*Firstly*, because it showed that diphtheria does not spread solely (as Bretonneau taught) by continuity of surface. *Secondly*, as accounting for death in *certain* cases previously inexplicable.

“From considerations flowing mainly from the discovery that the ‘continuity of surface’ theory was erroneous, I revived the doctrine that diphtheria was an essential fever, and hence the inutility of local treatment, at that time universally carried out, under the influence, and as a corollary to, the theory of its being a local disease.

“In Dr. Sanderson's essay, he seems to suggest that I had overlooked the fact of there being hyperoxidation in diphtheria. So far from ignoring it, it formed the basis of my views.

“Dr. Sanderson relates a case in which there was, with *albuminuria*, abnormal amount of renal excretion, and says that this case proves that my views are incorrect, and that in no case does insufficient elimination exist. I suggest that, *in the first place* (admitting all the facts of this case to be as he has stated), to draw the inference that, in no case can there be insufficient elimination, is to commit the logical error of reasoning from a particular to a universal. *In the second place*, admitting that the renal elimination going on during the height of the pyrexia was vastly greater than that of a period of health or convalescence when the ingesta were greater, proves nothing at all as to the adequacy of the elimination during the former period. The real question is this—‘*Was the elimination adequate to remove from the system the abnormally excessive quantity of effete material produced by the pyrexia?*’ To this Dr. Sanderson's comparative experiments give no answer. There are no scientific data in existence which can answer it. I admit that it is eminently desirable that these should be procured. But this is to be done, not by comparing





in the excretion of urea, and that the existence in the kidney of the condition implied by albumen and fibrinous casts in the urine, does not necessarily interfere with increase in the elimination of nitrogenous material. There is, therefore, no reason to apprehend the occurrence of uræmia as a consequence of the renal complication in diphtheria, this complication not being the cause of the blood-poisoning, but merely the index of its existence (*Brit. and For. Med.-Chir. Review*, Jan., 1860, p. 196).

**Phenomena and Symptoms.**—The *prodromata* which forebode an attack of diphtheria may be set down as general *malaise*, anorexia, slight fever, dysphagia, and glandular swelling. The symptoms generally supervene very gradually and insidiously; but feelings of depression, prostration, and muscular debility prevail, attended by headache, nausea, diarrhœa, and chilliness. There is a sense of stiffness about the neck and throat, and the drowsiness which often attends the accession of an attack of diphtheria may lead the patient to fancy he has caught a slight cold in the throat while indulging in a short sleep.

Dr. Jenner has grouped his cases of diphtheria into *six* varieties, as follows: (1.) *The mild form of diphtheria*; (2.) *The inflammatory form*; (3.) *The insidious form*; (4.) *The nasal form*; (5.) *The primary laryngeal form*; (6.) *The asthenic form*.

In the *mild form of diphtheria* the general symptoms and the local lesions are trifling, and no sequelæ follow. Febrile disturbance prevails to a slight degree; and there may be the least possible soreness of the throat on swallowing. No albumen occurs in the urine, and no nervous symptoms follow. Dr. Jenner is of opinion that many inflamed throats, when *diphtheria* is epidemic, have their origin in the *diphtheria miasm* (whatever that may be), just as many cases of *diarrhœa*, when *cholera* is epidemic, originate in the *cholera miasm*; and it is as difficult to say in some cases that an inflamed pharynx is not due to mild diphtheria as it is to say that a serious diarrhœa is not cholera.

In the *inflammatory form of diphtheria*, symptoms of severe *cynanche pharyngea* precede the exudation of lymph. There is redness, of a vivid or dusky hue, and swelling of the mucous membrane, covering the arches of the palate, the uvula, and the tonsils. The swelling is often considerable, from the effusion of serum into the sub-mucous tissue, which becomes of a jelly-like transparency and aspect. The pain in the act of swallowing is great, so that deglutition becomes impossible. The febrile disturbance may be extreme or moderate; and although the pulse is frequent, it soon becomes weak, and there is the sense of considerable prostration. In from twelve to forty-eight hours after the first symptoms of the throat affection supervene, a layer, more or less extensive, of tough lymph coats the inflamed surface, and death may follow from extension of the exudative process into the larynx or trachea. The urine may contain albumen, and sometimes the joints are swollen, hot, and tender.

The *insidious forms of diphtheria* are dangerous, because they seem sudden and unexpected. The general symptoms are not severe.



cific disease terminates between the eighth and fourteenth day of the illness (JENNER).

**Prognosis.**—However mild a case of diphtheria may appear to be, no case is unattended with danger. The great danger during the first week is from extension of the exudative process to the larynx; and the least laryngeal quality in the respiration heard at the bedside is suggestive of danger. Subsequently to the first week death is to be apprehended from exhaustion and loss of nervous energy. An extremely rapid and feeble pulse is of grave import; and a very infrequent pulse is of fatal significance. Vomiting is another unfavorable symptom, especially if it should recur many days in succession. Hemorrhages and albumen in the urine indicate blood change of great severity; and if the albumen is abundant, a fatal termination of the case may be expected. All the cases in which Dr. Jenner has known delirium to occur have ended fatally.

The danger in diphtheria seems to be in proportion to the youth of the patient. In the child, death is generally due to the extension of the disease to the larynx; after puberty it more often occurs from the general affection.

**Sequelæ.**—After the termination of the disease, symptoms of a very peculiar and characteristic kind are apt to supervene. The phenomena are referable to deranged innervation; and although their frequency and intensity are by no means invariably proportional to the severity of the primary disease, yet the more severe the case is, the more likely is nervous disorder to occur, and the more intense is it likely to prove. These consecutive phenomena do not appear at once. There is usually a brief period of convalescence between the disappearance of the primary and the appearance of the secondary phenomena of diphtheria. This period of temporary convalescence varies from a few days to a few weeks. The most alarming symptoms are referable to the heart. The frequency of its beats per minute begins to diminish, and a sense of languor supervenes, with tendency to vomiting. The heart's beats are found to be feeble, infrequent, and slow, and death supervenes from cessation of the heart's action (JENNER); or suddenly, from the deposition of fibrine within the heart, or in one of the great vessels (TANNER).

In other cases the paralysis is more widely spread, and the nervous symptoms more striking; although the nerve affections do not at once attain their maximum of intensity, but are progressive; and the progress of the paralysis, even in the same set of muscles, is seldom quite uniform. It is believed that the paralysis is due to a primary peripheral alteration of the nerves, which is propagated from the originally affected part to the spinal centre, much in the same way as in tetanus the irritation is transmitted from the wound (WEBER). If several sets of muscles are attacked, the faucial or pharyngeal are usually the set to suffer; and the impairment of function is very early betrayed by the condition of the voice, and by the act of swallowing, with loss of sensibility of the *velum pendulum palati* (TROUSSEAU). The sight is subsequently apt to become impaired; then the muscles of the tongue, the lips, and those of the



and family constitution (rather than any anti-hygienic conditions) favors its development and determines its progress (JENNER, GREENHOW, SANDERSON).

**Treatment.**—So long as there is heat of skin and firmness of pulse the physician ought to abstain from alcoholic stimulants, and rest contented by giving such saline medicines as exert a slight action on the skin and on the kidneys, or on both. *Acetate of ammonia* and *citrate of potash* are well suited for this purpose. The bowels should be opened freely by a dose of *calomel* and *jalap*; or by *calomel* and *colocynth* pill, followed in the inflammatory or *sthenic* forms of the disease by a saline aperient—*e. g.*, *sulphate of magnesia* in the *infusion of roses*.

The throat affection should be treated with warm fomentations externally, and by the inhalation of water vapor with acetic acid. A wine-glassful of vinegar to a pint of water is a good proportion (JENNER), and an inhaler should be used, as mentioned at page 317, under scarlet fever. Dr. Jenner recommends Squire's inhaler as the best. A lead gargle may be of service, composed of one fluid drachm of the solution of diacetate of lead in eight ounces of rose-water; but gargles must not be persisted in if pain is caused by their use. The temperature of the room in which the patient is confined to bed ought to be kept at 68° Fahr., and its atmosphere made moist by the steam from a kettle with a long spout constantly boiling on the fire. If the patient can be enveloped in a warm moist atmosphere, so much the better; and this may be done by making a tent with blankets over the bed, and, by the aid of a spirit-lamp, a tin kettle of boiling water may be maintained at the boiling-point, and its steam thus made to envelop the patient.

If feebleness of pulse supervene, if the redness of the throat assume a dusky hue, if the sense of general weakness become extreme, wine in large doses frequently repeated is required. Six or eight ounces of port or sherry during the day for an adult may be given from the first, with as good a diet as the stomach can digest. During the course of the disease, much larger quantities of wine and even brandy may be necessary; but the quantity of stimulants must be regulated by the habits and age of the patient. A child of three years of age may take with advantage one or two drachms of brandy every hour—*i. e.*, from three to five ounces of brandy during the twenty-four hours (JENNER). Under all circumstances efficient daily action of the bowels must be secured, and the urinary and intestinal secretions should be examined daily.

If blood or albumen appear in the urine, diuretics are contraindicated. Mustard poultices, warm linseed-meal poultices, or the warm wet sheet, as recommended by Dr. Huss in typhoid fever, and referred to at page 387, may be applied to the loins under these circumstances. *Tincture of the perchloride of iron* has been recommended by Dr. Hislop, of Birmingham; and it may be advantageously combined with quinine in the following formula (TANNER):

R. Quinæ Sulphatis, gr. ij; Acidi Hydrochlorici diluti, ℥x; Tincturæ Ferri Perchloridi, ℥xv; Infusi Calumbæ, ʒj; *misce. Fiat haustus, omnibus sextis horis sumendus.*



plated by an opening in the windpipe is the prevention of death by suffocation. By so averting death, time is gained for the general disease to run its course (JENNER).

### CROUP.

LATIN, *Angina trachealis*; FRENCH, *Croup*; GERMAN, *Croup*—Syn., *Häutige Bräune*; ITALIAN, *Laringitide membranacea*.

**Definition.**—A specific disease, accompanied by the exudation of an albuminous material upon the mucous membrane of the epiglottis, glottis, larynx, or trachea, and sometimes over all of these parts, indicated by accelerated, difficult, wheezing, or shrill respiration; short, dry, constant, barking cough; voice altered by hoarseness, with spasm of the interior laryngeal muscles, and pain and constriction above the sternum; frequently followed towards the close of the disease by expectoration of a membranous albuminous substance, or even of a cylindrical cast of some portion of the breathing-tube. The disease occurs in children, and may terminate fatally either in suffocation or exhaustion of the vital powers.

**Pathology and History.**—It has often excited much surprise that a disease so distinctly marked in its symptoms should not have been accurately described before the middle of the eighteenth century, when Dr. Francis Home published a treatise on the *suffocatio stridula* or *croup*, in 1765, as it was observed in Leith, Musselburgh, and the vicinity of Edinburgh. It has been described under the name of *cynanche trachealis*; and Dr. Farr has proposed for it the name of "*trachealia*" in scientific nosological nomenclature.

Before the time of Dr. Home, however, there is reason to believe that the disease was confounded with other affections of the throat and breast resulting simply from exposure to cold. It was certainly also described and distinguished by Martin Shisi, in 1749, at Cremona, and by Starr, of Liskeard, in Cornwall, in the same year (*Phil. Trans.*, 1750). Many physicians have described the disease since that time, and none with more minuteness than Dr. Cheyne, of Leith, who observed it for several years, and illustrated its pathology by careful dissections.

The most remarkable pathological phenomena of croup are to be observed in the exudative process which attends the inflammation in the windpipe, and the formation of a false membrane, almost peculiar to children, but sometimes seen in adults. When death takes place after an illness of four or five days, the windpipe is found to be lined with a white or gray substance. The membranes thus formed vary much in thickness and consistency. Some are so thin that the mucous membrane is readily seen through them, while others are many lines in thickness, exceeding even that of the mucous membrane itself, and consequently opaque. With respect to their consistency, some are so little coherent that they are almost diffuent, while others can be detached for a considerable extent without rupturing. The false membrane, though occasionally only partial, yet more commonly embraces the entire circumference of the larynx,





the fluid when first exuded from the inflamed vessels, as may be ascertained by the administration, upon the approach of the symptoms, of a powerful emetic, which will bring away this fluid before it has concreted into a membrane; these globules generally attracting each other, and appearing like bloodvessels, as the albuminous matter coagulates on the inflamed surface; (c.) That the membranous substance is detached in the advanced stages of the disease, by the secretion from the excited mucous follicles, of a more fluid and a less coagulable matter, which is poured out between it and the mucous coat; and, as this secretion of the mucous *cryptæ* becomes more and more copious, the albuminous membrane is the more fully separated, and ultimately excreted if the vital powers of the respiratory organ and of the system be sufficient to accomplish it; (d.) That subacute or slight inflammatory action may be inferred as having existed, in connection with an increased proportion of fibro-albuminous matter in the blood, whenever we find the croupal productions in the air-passages; but that these are not the only morbid conditions constituting the disease; (e.) That in conjunction with the foregoing—sometimes only with the former of these in a slight degree—there is always present, chiefly in the developed and advanced stages, much spasmodic action of the muscles of the larynx, and of the transverse fibres of the membranous part of the trachea, which, whilst it tends to loosen the attachment of the false membrane, diminishes, or momentarily shuts, the canal (of the larynx) through which the air presses into the lungs; (f.) That inflammatory action may exist in the trachea, and the exudation of albuminous matter may be going on for a considerable time before they are suspected—the accession of the spasmodic symptoms being often the first intimation of the disease; and these, with the effects of the pre-existing inflammation, give rise to the phenomena characterizing the sudden seizure; (g.) That the modifications of croup may be referred to the varying degree and activity of the inflammatory action, the quantity, the fluidity, or plasticity of the exuded matter, the severity of spasmodic action, and to the predominance of either of these over the others in particular cases, owing to the habit of body, temperament, and treatment of the patient, &c.; (h.) That the muco-purulent secretion, which often accompanies or follows the detachment and discharge of the concrete or membranous matters, is the product of the consecutively excited and slightly inflamed state of the mucous follicles, the secretion of which acts so beneficially in detaching the false membrane; (i.) That a fatal issue is not caused merely by the quantity of the croupal productions accumulated in the larynx and trachea, but by the spasm, and the necessary results of interrupted respiration and circulation through the lungs; (k.) That the partial detachment of fragments of membrane, particularly when they become entangled in the larynx, may excite severe, dangerous, or even fatal spasm of this part, according to its intensity relatively to the vital powers of the patient; and that this occurrence is most to be apprehended in the complicated states of the malady where the inflammatory action, with its characteristic exudation, spreads from the fauces and pharynx to the larynx and



exudation grows upon the inner surface of the air-passages, constituting the false membranes already described. The first form seems to be the one common in America, of which not more than *one* in *fifty* dies. The latter is the more common European form, of which the deaths used to be *four* out of *five*, and still are about *a half*. About one child in twelve deaths of children dies from this disease; and the ratio borne by croup to 1000 deaths from all causes, in 1854, was as 9.249.

**Symptoms and Course.**—The mildest form of croup differs from an ordinary catarrh only in the addition of spasmodic symptoms; but this form may run into the more severe form, so that it is not possible to determine, in the first instance, which form the disease may ultimately assume.

The catarrhal croup of Dr. Wood embraces the spasmodic as well as the catarrhal croup of Dr. Copland. Spasmodic action of the laryngeal muscles is, however, common to both, and is characteristic; but the inflammation and exudation is not in general more severe than that which attends a common catarrh.

The disease may be ushered in by sore throat, by catarrhal symptoms, or by a short dry cough; or it may occur *per se*, and without the general health being sensibly impaired. In either case the attack commonly takes place during the night, the sleep of the child, which was perhaps more or less agitated, being interrupted by fits of *hoarse* coughing. These become more frequent, the respiration more difficult, and marked by a peculiar wheezing, which has been described as like the sound of an inspiration forcibly made with a piece of muslin before the mouth, or like to the sound of air passing through a brazen tube. The little patient also feels a sense of restriction about the throat, as shown by carrying the hand often to it, and grasping the larynx. After the paroxysm has lasted some hours, there is an interval of ease, which perhaps lasts for some hours.

By the end of the second or third day, sometimes sooner, the tongue becomes white, the heat of the body increased, the pulse frequent, the face flushed, and the countenance distressed. From this point the disease now rapidly advances, the croupy sound attains its height, and Dr. Home describes it as "*vox instar cantus galli*;" others have compared it to the noise which a fowl makes when caught in the hand; while the child often puts its fingers into its mouth, as if to pull away something which obstructs the passage.

As the disease draws towards a close the paroxysms become more frequent, the cough more severe, the pulse more rapid, suffocation more imminent, and the extremities cold and livid. The final close of the disease is often by convulsions, sometimes almost tetanic; and Dr. Ferrier once was present when the struggle was so violent that after death the corpse, in a great measure, rested on the occiput and on the heels.

Often, however, the symptoms are much more moderate; although it not unfrequently happens that symptoms of the severer form come on, indicated by a huskiness of the voice, till no sound can be



croup, opinions are very much divided as to the nature of the epidemic influence, and whether or not the disease is contagious or infectious.

Age has, perhaps, the greatest influence in predisposing to the disease, and, while rare in adults, it is seldom seen in early infancy. It is most prevalent between the *first* and *seventh* years of life. According to the experience of Dr. Wood, the disease appears to run in families; and vigorous fleshy children, with rosy complexions, are frequently those who suffer most.

**Prognosis.**—"Is never better than doubtful." It is to be determined from the violence of the local symptoms, and the frequency of the paroxysms, rather than from the constitutional symptoms. Children, however, seized with croup are said to recover in a smaller proportion in this country than in America. Death tends to occur by *apnœa*.

**Treatment.**—*Every case of croup demands the most active, efficient, and energetic treatment.* When the croup in children commences in the larynx, its course is so rapid and so fatal that the measures for its suppression must be early. Bleeding, and especially local bleeding, should be employed, and in most cases to a considerable extent (an ounce of blood for every year of age); and two to twelve leeches, according to the age of the patient, should be applied over the larynx. After these have fallen off, the bleeding should be encouraged by the application of a linseed poultice to the throat. This first bleeding often gives great relief, and sometimes averts the disease; but if not, the leeches, after a few hours, may be repeated. As soon as some relief is obtained, a blister should be applied along the lateral aspect of the neck on each side, and not over the trachea; and after that is removed, the part should be dressed with strong mercurial ointment. In addition to bleeding and blistering, many practitioners prescribe *emetics*; first, because their emetic effects, and the large evacuations they produce, favor the resolution of the inflammation; and again, because the effort of vomiting may be the means of detaching and of expelling the false membrane, should it have formed. If relief does not ensue on the action of the emetic, Dr. Cheyne recommends *two, three, or four* grains of calomel, with *two or three* grains of James's powder, to be given at short intervals every two or three hours; and a dose of castor oil is to be given occasionally till the full effect of the calomel as a purgative is obtained. Green fecal stools, like chopped spinach, are characteristic of this result.

Bleeding, blistering, and mercury, although the rule of treatment in idiopathic infantine croup, are, for the most part, entirely inefficient in those cases in which the affection begins in the fauces, as in the case of many epidemics, and especially after scarlatina. In these cases the best treatment, if the false membrane be not already formed, is to relieve the throat by the application of a few leeches, as in scarlet fever, and then to support the little patient with a moderate quantity of wine diluted with water. If the false membrane has formed, perhaps an emetic affords the only chance of relieving the patient; and, indeed, so soon as croupy cough and



neau saved six. Perhaps the experience of the profession, generally, is equally discordant on this point at this moment; for those who operate early contend they save some portion of their patients, while those who wait till a case is advanced, and beyond medical treatment, before they resort to this measure, for the most part lose all their patients. The evidence, however, is daily accumulating which shows that tracheotomy ought to be resorted to much oftener, as a remedy for croup, than it has hitherto been, and that *at a much earlier period in the disease,—not as a last resource, when death from asphyxia appears imminent, and after treatment of the most depressing kind.* That this is the secret of success in France and in this country is shown by the experience of able physicians and good surgeons, of whom the names of M. Trousseau, the late Mr. Jones, of Jersey, Mr. Henry Smith and Dr. Fuller, of London, the late Dr. Cruickshank, of Dalmellington, in Scotland, and Mr. Spence, Professor of Surgery in the University of Edinburgh, Dr. George Buchanan, in Glasgow, and Professor Roser, of Tübingen, may be stated as authorities by experience. In country districts the performance of tracheotomy in a case of croup is almost imperatively called for in the majority of cases, *if some symptoms of amelioration do not follow the steady use of bleeding, emetics, the warm bath, and calomel purgation, pursued for twelve or sixteen hours.* I know that, in a wild country district of Scotland, where croup was very common and fatal, the late Dr. Cruickshank saved eight out of eleven cases during two years. A valuable paper by Mr. Smith, in *The Medical Times and Gazette*, 26th January, 1856; another by the late Mr. Jones, of Jersey, on the 8th November of that year; and, lastly, a paper by Dr. Conway Evans, in the *Edinburgh Medical Journal* for January and May, 1860, go to support the same conclusion,—namely, that an earlier introduction of air, by the operation of tracheotomy, for croup, would not only give a larger percentage of recoveries in this country, but would place the operation in the same favorable light in which it is now regarded in Paris and other parts of France. Tracheotomy in croup is undoubtedly gaining ground; and it cannot be denied that children perish in the first instance almost always from suffocation. Tracheotomy is therefore indicated in croup (as in diphtheria) as soon as there are urgent symptoms of obstruction of the glottis. When the respiration is so impeded that the demand for oxygen is only satisfied by difficult forced respirations, dreadful anguish is depicted on the reddened countenance covered with sweat; there is extreme restlessness; the patient tosses from side to side, gets out of bed one minute and into it the next, clutching spasmodically at those around him, as if seeking everywhere for help. *This is the proper period for the operation of tracheotomy in croup,—the time when success may be expected (ROSER).* Should the operation be longer delayed, symptoms of asphyxia appear, overloading of the blood with carbon ensues, the face suddenly becomes blue, with fixed and staring eyes, convulsive exertions are made, and anxious struggles for breath follow the stage of suffocative agony. In some cases the symptoms of asphyxia come on more slowly and are apt to make considerable progress





*the mucous membrane of the large intestines, with the intertubular connective tissue, are the chief seats of the local lesion, which sometimes extends into the small intestine beyond the ileo-colic valve ; as in cases in which scorbutus is a predisposing cause.*

**Historical Notice, Pathology, and Morbid Anatomy.**—Dysentery is a disease which varies considerably in different countries and localities ; and sometimes in apparent accordance with the exciting cause. Sporadic cases, which now and then occur in our large towns, are not generally so violent, and are less fatal than the epidemic cases, and those which occur in tropical climates. The effects on the constitution are no less varied and severe.

Dysentery has at all times proved one of the most severe scourges of our fleets on foreign stations, of our armies in the field, and during campaigns, even in temperate regions. It is sometimes so prevalent that it exceeds the number of sick from all other diseases put together. It has followed the tracks of all the great armies which have traversed Europe during the Continental wars of the past 200 years. It helped to destroy the British army in Holland in 1748. It decimated the French, Prussian, and Austrian armies in 1792. It was a chief cause of death in the ill-fated Walcheren expedition in 1809. It cut down the garrison of Mantua in 1811 and 1812. Sir James McGrigor records how fatal the disease was in the Peninsular campaigns ; and we know how disastrous it was to our troops during the first winter they passed in the Crimea, in 1854. In the words of Sir Ranald Martin, “It is the disease of the famished garrisons of besieged towns, of barren encampments, and of fleets navigating tropical seas, where fruits and vegetables cannot be procured. During the Peninsular war, the first Burmese war, and the late war with Russia, *dysentery* was one of the most prevalent and fatal diseases which reduced the strength of the armies.”

That it is a dangerous and frequent disease throughout our inter-tropical possessions, the tabular statements on the next page, furnished by Sir Alexander Tulloch to Sir Ranald Martin, and by Dr. Joseph Ewart, of Calcutta, sufficiently testify.

In England, generally, however, *dysentery*, as a cause of death, has been decreasing since 1852, although about 200 years ago it was one of the most prevalent and fatal diseases of London. Yet still, although the disease is less violent and less fatal (for as a cause of *death* it has remarkably diminished during the past ten or twelve years), and although the unfavorable hygienic conditions which were wont to bring about dysentery no longer exist, the active endemic conditions which favor, promote, or are congenial to its development, are only dormant, and not eradicated. The disease, therefore, is still sometimes brought about just as in the days of Sydenham or Willis. In no respect, however, do we find that the dysentery of this time differs essentially from the description given by Sydenham more than a hundred and thirty years ago. When we look, therefore, to the history of the disease, and to the nature of its lesions—to its reappearance from time to time among us, with the same identical characters—there are strong grounds for



believing that there is something specific in the nature of the poison which produces dysentery, just as specific as that of *small-pox*, *typhus fever*, *typhoid fever*, *yellow fever*, *scarlatina*, *ague*, or *diphtheria*. But besides the specific identity of the disease, as it now exists, with the disease of former times, there is another point of view from which the history of the pathology of dysentery is especially instructive. It is this: Like all diseases which have been at the same time epidemic and severe, it has been the subject of discussions as frequent and as varied as its ravages have been severe; and one single description of the disease will not do for a record of the characteristics of all epidemics. Most minute descriptions of the state of the intestines in dysentery have been given by many writers; but, as Dr. Copland justly observes, from his extensive experience, "Dysentery is neither so simple in its nature, nor so unvarying in its seat and form, as most recent writers in this country have stated;" and "that writer will but imperfectly perform his duty who, in giving a history of a most prevalent and dangerous malady, confines himself to the particular form it has assumed during a few seasons, within the single locality, or the small circle of which he is the centre, and argues that it is always as he has observed it."

Dysentery is, moreover, a most formidable disease, on account of its oftentimes insidious nature, from its tendency to recur, and from the after-influences it exerts on particular organs and on the system at large. For these reasons almost all writers on the diseases prevalent in tropical climates place dysentery at the top of the list of severe affections, and refer to it as the cause and origin of many of those chronic and intractable abdominal diseases which so often afflict Europeans resident in tropical climates; and which entail most varied forms of impaired health when they return to European climates.

The morbid anatomy of dysentery has not been described with uniform distinctness, and the anatomical descriptions have, in general, been extremely vague. Medical science has not yet finally settled many points in the pathology of the disease; consequently, the doctrines as to treatment are somewhat uncertain, while the means of prevention are not less imperfectly defined. It has been usual to describe cases of *dysentery* as being either *acute* or *chronic*; but there are also cases belonging to a third class, which may be termed *complex*.

**Acute Cases of Dysentery.**—In this form the specific lesion in the form of inflammatory action does not confine itself to the tissues of the mucous membrane only. The serous covering of the intestines, or even such solid viscera as the liver, spleen, kidneys, are involved in a disease-process. Ulceration or sloughing of large portions of mucous membrane and exudation go on together, and there may be very little corresponding fever at all commensurate with the severity of the lesions, so that while the disease is acute, it is at the same time, in many instances, of a masked and almost latent nature. Death frequently takes place within the first ten or twelve days in such cases; but the disease may terminate gradually and spontaneously, or as the result of appropriate treatment, by the end of the third or fourth week. On the other hand, the disease may



and in the severer forms, the mucous membrane becomes gelatinous, and is easily separable, or it passes into a state of sphacelus, black, friable, and offensive. All these observers regard ulceration as having no essential part in the disease-process which constitutes dysentery, and as being of very rare occurrence.

Some of the writers who have described the tropical forms of the disease have been still less distinct as to the details of its morbid anatomy. For example, Twining seems to have followed Chomel in considering the lesion to be a simple inflammation of the mucous coat; and Annesley is in a great measure unintelligible as to the points of morbid anatomy which he describes. It was not till Dr. Parkes published his minute and admirable description of the morbid anatomy of dysentery, as he saw it in India, that we had anything definite on the subject as regards the tropical forms of the disease. He not only showed the very early implication of the glandular apparatus of the great intestine in dysenteric inflammation, but he established the fact, so far as his cases went, that, while ulceration occurs with great rapidity, a case never presents true dysenteric symptoms without ulceration being present. At Moulmein, in India, he investigated, in 1843-44, cases of dysentery in Europeans to the number of fifty, and in Asiatics to the number of twenty. He concluded from these observations that: (1.) Certain alterations in the glands of the mucous membrane of the large intestine, and sometimes of the ileum, constitute the earliest lesion in dysentery. (2.) That in all cases, when not too far advanced, the mucous membrane presented the appearance of numerous whitish round elevations, of a size varying from a millet-seed to a size so minute that a lens only can show the lesion. These elevations were hard, and being pierced, gave forth a white excretion. Many of these had a black speck in the centre, and were surrounded by a vascular circle. (3.) He noticed that exudation sometimes occurred in points beneath the mucous surface; that such points of exudation had a white appearance, with contents similar to those of the solitary glands. The mucous membrane over these points could be easily rubbed off, leaving an ulcer (Parkes *On the Dysentery and Hepatitis of India*).

The observations of Dr. Parkes were thus opposed to the views just stated, and led to extended investigation, by which such contradictions may be reconciled; and it was determined that differences of climate do not cause any essential difference in the structural changes which accompany dysentery. The observations of Drs. Craigie and Abercrombie in Scotland, in 1837, prove this; and also those of the late Dr. Baly, in 1847, as regards England. Drs. Cheyne, Graves, and Mayne have demonstrated the same fact as to Ireland. By the records of epidemic dysentery at Prague and elsewhere, as described by Dr. Finger and others, the observation holds true as regards the dysentery of Europe generally; and by comparing these records with the well-recorded cases of those who have seen the disease in the tropics, both in civil and in military life, it will be seen that the true dysentery of tropical and temperate climates does not differ *as to its anatomical signs* in any essential particular.

The descriptions of the disease in our own country, as given by



In this country it is believed that the lesion in dysentery is confined, for the most part, to the colon and rectum; but that in tropical dysentery the whole course of the colon, and sometimes a considerable portion of the small intestines, are implicated. But, except when the case is associated with *scorbutus*, the small intestines are not involved. Lesions so extensive, while they are common in India, are rare in this country; yet they do occur, and are not uncommon in the south of Europe, in Turkey, and the coasts of the Mediterranean. Therefore, as regards the extent of the lesion, there is no constant or distinctive characteristic between tropical dysentery and the dysentery of more temperate climates.

In both regions the anatomical changes comprehend redness of the mucous membrane, preceding further changes; loss of the substance of the mucous glands by pulpy softening of tissue, sloughing, or ulceration; the detachment of diphtheritic casts of the intestine, or sloughs of tissue.

In describing the morbid anatomy of dysentery the reader is referred to the nomenclature of the gland structures, given in a footnote at page 364 of the present volume. The structure of the colon in the healthy state differs in many important particulars from that of the small intestine. It is remarkable for the absence of folds and villi, and for the presence of more or less dilated sacculi, which give form and shape to the excrement. The minute tubular glands are thicker in proportion to their length, compared with those of the small intestine; and the intertubular connective tissue is considerable,—a structure which takes an important share in the lesions of dysentery. These tubular glands are lined by columnar, cylindrical, and transition forms of epithelium; and the solitary lenticular glands are sometimes closed vesicles (ALLEN THOMSON, PARKES, BALY), and sometimes open follicles. When closed they are not visible; but if distended, they may be seen with a lens; and when open, a dark depressed point marks the separation in the tubular gland structure which leads to the open follicle. The tubular glands radiate round this spot, which corresponds to a depression indicating the empty vesicle below. These solitary vesicles have thick walls, and are said to be more abundant in the cœcum and rectum than in any other part of the great intestine. This statement leads to the question which has been mooted in relation to these solitary gland lesions, namely,—“Are these lesions of the so-called solitary glands really due to the enlargement of previously existing solitary glands or their germs? or, Are they new formations altogether?”

A similar question is at issue regarding the granulations on the eyelids and conjunctivæ, associated with purulent ophthalmia (STROMAYER, FRANK, MARSTON). In this disease we have *new formations* of vesicular-like granulations, as well as enlarged follicles; but these are more numerous than the glands have ever been seen to exist in the healthy state. Observations somewhat similar have been made regarding the vesicular glands of the stomach (HANDFIELD JONES). It may be, therefore, that not a few of the “tubercle nodules,” the “pustules,” the “small-pox-like elevations,” and what we call solitary or lenticular glands, are in reality new formations altogether,





soften and to be cast off as an exuvium or slough, exposing the submucous connective tissue or the muscular coat of the intestine. It is the mucous membrane of the great intestine, and especially of the rectum and lower portion of the colon, which is the seat of these characteristic lesions in dysentery. The exudative process is generally diffuse, involving the whole of the tissues of the mucous membrane. In some cases, however, it is seated in the solitary glands, in the first instance, and neighboring mucous tubular glands.

In the scorbutic form of dysentery, or in dysentery occurring in persons whose nervous or vital powers are feeble, or below *par*, as in the aged, infirm, or in the paralysis of the insane (conditions in some respects similar in their influence to *scorbutus*), a diphtheritic exudation covers to a considerable thickness not only the mucous surface of the colon, but (as an almost constant and pathognomonic morbid sign) the same lesion is seen covering the mucous surface of the small intestine, extending upwards from the ileo-colic valve; and, as Dr. Parkes has noticed, this exudation grows or is laid down especially in the course of the bloodvessels ramifying from the mesenteric attachment transversely across the surface of the gut, and occupying especially the prominences of transverse rugæ. This form of dysentery prevailed to a great extent among the soldiers who died at Scutari during the period of the Russian war when *scorbutus* prevailed. Some of the cases recorded by Dr. Davis, in his admirable description of the dysentery so fatal to the British troops in the famous Walcheren expedition, were also of this nature. Dr. Finger, of Prague, and Dr. Maine, of Dublin, and Dr. Baly, have recorded similar cases; but one of the best accounts of this form of dysentery is that which Dr. Clouston records as having prevailed in the Cumberland and Westmoreland Asylum, and which he believed to have been caused by the effluvia from a field irrigated with sewage. The regular diet of the Asylum inmates consisted of 24 ounces of animal food, 14 pints of milk, 16 ounces of suet dumpling, 7 pints of oatmeal porridge, 78 ounces of bread, and 7 pints of tea per week (*Med. Times*, June 3, 1865). No fresh vegetable diet seems to have been provided for; and diarrhœa does not seem to have been uncommon.

In typical cases of the outbreak described by Dr. Clouston—

“All the abdominal organs would be found healthy until the small intestine was examined. This, too, would be normal up to within five or six feet of the cœcum. The mucous membrane would then begin to appear reddened in small spots or rings round the gut. Six inches farther down the redness would be universal, and the membrane would begin to be thickened and corrugated into folds, like small *valvulæ conniventes*. A few inches farther down, a yellowish, dirty-looking deposit would be seen over the mucous membrane in rings, very thin where it began, but gradually becoming thicker and more continuous till near the cœcum it would be one-eighth of an inch in thickness. The swelling of the mucous membrane would also increase downwards, and the folds running across the gut would become more prominent. These, with their coating of deposit, made the inside of the bowel look like a series of thick transverse ridges,



The exuded mucinous material, in its more recent state, forms a layer, which varies from a thin but opaque membrane to three or four lines in thickness, of homogeneous substance, tolerably consistent, and capable of being detached and raised in flakes from the subjacent mucous surface. During the earlier stages of the disease the surface of the mucous membrane appears unchanged below, except, perhaps, by the existence of a little increase of vascularity. The color of the exuded matter may be uniform, or red, white, or pink in patches, and discolored in some instances by intestinal gases, the biliary secretion, or by the admixture of blood, and the changes consequent thereon. The most common appearances in severe cases is that of a dark olive-green, passing into a bluish-black. The surface of the exudation may be uniform, or the whole aspect may be mammillated, with here and there a *mammillation* projecting greatly above the others in a fungating mass, surrounded by dark fissures in the exudation. These fungating masses are soft towards their centres, with numerous red vascular points here and there on the surface. A section through the mass shows the base thickened and firm.

The dysenteric process, as seen after death, is generally found to have advanced farther in one part of the intestine than in another; usually, it may be stated to have been farther advanced in the *rectum* than in the *descending colon*, and farther in that part than towards the head of the large intestine. In well-marked and extreme cases the entire mucous surface, from the *caput cæcum* to the *rectum*, may be seen to present all the possible stages of the dysenteric process. Three stages can in general be distinguished, namely,—(1.) Ulceration of the exudation and mucous membrane more or less advanced towards the *rectal end* of the great intestine; (2.) Exudation in various forms towards the *middle* of the *colon* upwards from the *rectum*; (3.) The exudative process visible microscopically in the tubular glands, and sometimes also obvious to unaided vision in the solitary vesicular glands of the great intestine towards the *caput cæcum*.

One of the best descriptions of the morbid anatomy of dysentery in the English language has been given by the late Dr. Baly in his *Gulstonian Lectures* for 1847. He describes *three* different forms of lesions as seen by him amongst convicts at the Millbank Prison; and these three forms he believed to correspond with three degrees of severity of symptoms during life,—(1.) He recognized a swollen condition of the solitary glands, forming round prominences on the surface of the mucous membrane, of various sizes. In color these were pale, or red round the base, and dotted at the summit with a vascular spot. These appearances would occur about the eighteenth or twentieth day. At an earlier period the congestion round the glands would be more intense; while at a later period the summits of the prominences would become disorganized. Minute yellow sloughs subsequently form, which, becoming detached, leave an ulcer previously occupied by the gland. The mucous membrane around participates in the process. It is red, tumid, and covered with an aphthous layer of lymph, to the extent of one or two inches



offensive smell. [Dr. Chuckerbutty\* found the sediment, after washing by the plan of Goodeve, to consist of a certain mucus,—ropy, gelatinous, branny, or thready; shreddy, or granular lymph; laudable, ichorous, or scrofulous pus; fæces in various degrees of consistence; and sloughs.] He observed that patches of membrane, half an inch or an inch or more in size, are cast off as sloughs. These exuviæ are thin, membranous, and sometimes infiltrated with pus; or they are thick and of a yellowish-brown color. It is not till after the *eighth* or *twelfth* day of the disease that such sloughs are cast off. In these respects they may be considered similar to those cast off from Peyer's patches in typhoid fever. After these shreds are cast off the symptoms diminish, and the patient often gets well rapidly.

[The conditions indicated by the various kind of sloughs are, according to Dr. Chuckerbutty, as follows:

CHARACTER OF SLOUGHS.	CONDITIONS DENOTED.
Ecchymosed, . . . . .	Abraded or minute ulcers; intestinal apoplexy.
Compact gray or light-yellow, . . . . .	Acute phlegmonous dysentery.
Thick pus—infiltrated mucus, . . . . .	Erysipelatous dysentery.
Ragged, . . . . .	Gangrene
Dark olive, . . . . .	Secondary gangrene of mucous coat.
Thin black, plain, or tubular, . . . . .	Primary gangrene of mucous coat.
Shreddy, . . . . .	Gangrene in either mucous or cellular coat.
Molecular, or putrilage, . . . . .	Disintegration of tissue.
Flaky epithelial, . . . . .	Commencing gangrene of mucous membrane.
Shaggy, . . . . .	Violent inflammatory action.
Free filamentous (simple), . . . . .	Primary gangrene in submucous tissue.
“ (pus-infiltrated), . . . . .	Submucous cellulitis.
Ring-shaped, . . . . .	Ring-shaped ulceration in mucous folds.
Discoid, . . . . .	Circular ulcers in ecchymosed patches.]

During the shedding of the shreds the patients are much griped, and they pass with straining the sanguinolent masses, or slimy mucus in small quantities, and generally without fecal matter, fifteen or even twenty times a day. Then a period of cure and improvement supervenes, with diminution of the stools or of the fecal discharges,—not simply by resolution, but a termination by elimination of the specific sloughs or lesions which have formed in the course of the disease. In this respect the phenomena seem analogous to what occurs in typhoid fever. When these membranous flakes are not shed, but retained and ultimately separated in large pieces, there is considerable danger attending the process. Discharges of blood and fatal hemorrhages may ensue. Morehead records eight cases of this kind in India, and four of them were fatal, of whom one died from hemorrhage. To account for this hemorrhage, it has been observed that changes go on between the intertubular connective tissue and the substance of this exudation,

---

\* [Cases Illustrative of the Pathology of Dysentery, with Remarks. By S. G. Chuckerbutty, Calcutta, 1865.]



The *circular* ulcers, for the most part, originate in the solitary glands (PARKES, BALY), or in circular patches of tubes (MOREHEAD), similarly to the stomach ulceration, as described by Drs. Handfield Jones and Brinton; or such circular ulcers may result from both, as when a solitary gland is destroyed it carries with its destruction some of the adjacent tubes. In such cases the colon presents prominent little masses, about the size of a pea, which burst readily on pressure, and give forth fluid contents like pus. Such abscesses may open spontaneously upon the mucous surface through the short canal leading from the vesicular gland (now an abscess), imbedded in the submucous tissue, and between the tubular glands. They undermine the tubular gland-substance, and carry off shreds or patches of the surrounding tissue. They may thus be seen in all stages, and sometimes almost symmetrically arranged in a double row along the colon (BLEEKER, MOREHEAD). Many of these little abscess cavities are also formed below patches of thick exudation [HASPEL].

The *transverse ulcers* are due to the transverse arrangement of folds, on which the exudation and textures ulcerate, as already described, and I have known the transverse *rupturing* of very thick exudation mistaken at post-mortem examinations for ulceration, on seeing the raw vascular surface of the tissue exposed below at the bottom of the rent.

Microscopically the exudation in its most recent condition may be seen to be composed of fine germs and nuclei, with elongated nuclear cells. It appears to be chiefly exuded into the follicular and tubular apparatus of the mucous membrane, and gradually accumulating there, is pushed upwards to the mucous surface, which it finally overspreads as a whitish coat, coherent and uniform, susceptible of vascular organization, and tending to ulcerate.

**Anatomy of the Tissues in Chronic Dysentery.**—In the true chronic form of dysentery the exudation already noticed undergoes various changes. It may be thrown off from the mucous surface altogether, leaving that surface bare and raw-looking, as if ulcerated; but a close inspection will show that the surface is entire and highly vascular. If it is not thrown off, it may undergo a considerable amount of organization; after which it appears that a process of ulceration may be established upon its surface, just as in any other soft tissue. This ulcerative process may extend through the whole exudation, even to the surface of the mucous membrane, which it may penetrate also, and involve the tissues of the intestine in the ulcerative process close to the peritoneal coat. Perforation of the peritoneum is by no means uncommon.

In the chronic forms of dysentery there is a very constant morbid change to be observed, consisting in the deposit of black granular matter on some parts of the mucous membrane. It may be regarded as the result of excessive vascular action, and of subsequent changes in the extravasated blood, elements which mark the site of the melanic spot (pigmentary degeneration; see page 121).

The sigmoid flexure of the colon is perhaps the most frequently and the most extensively diseased, and the lesion is most expressed towards the rectum. In very severe cases the exudation extends





edges so sharp, clean, and perpendicular, that they appear as if made with a punch. In other cases the tubular glands, as well as the solitary and aggregate glands of Peyer, have shown various stages of morbid action. The absorbent mesenteric glands are rarely affected (BALY); but except in cases of secondary hepatic abscess, they were found enlarged and inflamed in all cases of Indian dysentery (PARKES), and also in the dysentery associated with *scorbutus*.

By far the most common condition, however, in chronic cases of *dysentery* especially, is that which is due to atrophy of the mucous membrane. As an atrophic change, it may be ascribed to the general wasting (marasmic) processes which take place to a great extent throughout the system in cases of *chronic dysentery*. In this complex state the mucous membrane of the *small intestine* appears pale, thin, and worn,—a condition which pervades the greater part of the alimentary canal, and which is especially made manifest in the living as well as in the dead by the condition of the mucous membrane of the mouth. On turning down the lips, the mucous glands are seen distinctly projecting through the thin pale labial and buccal mucous membrane. When such cases are examined after death, the structure of the solitary glands and of Peyer's patches are found to be degenerated and wasted; no gland-cells are to be seen, and their place is supplied by fibroid tissue, with some vascular injection round the reticulated spaces. In other instances a deposit of black pigment surrounds the locality of the glands, which indicates the long-continued process of vascular action previous to their atrophy. Associated with this general atrophic state, some gland-patches may be observed in an apparently opposite state—that is, distended, and sometimes engorged; but, on examination, their contents appear to be undergoing a molecular, melanotic, and generally fatty degeneration, probably preparatory to complete evacuation and destruction of the gland-element. These two apparently opposite conditions, coexisting in the same cases, appear to indicate that the one condition is but the antecedent of the other; and that the atrophy and degeneration is the last result of a series of morbid processes commencing in the engorged gland-cavities.

In parts of the mucous tissue which exhibited the opposite conditions of extreme hypertrophy and extreme atrophy, the specific gravity of the former indicated 1.046, while the thin and wasted part of the intestine indicated a specific gravity of 1.036 to 1.030.

There is now abundance of evidence to show that, in some endemic cases, or in epidemics of *dysentery* in some places, there is a tendency to the secondary affections of organs or parts, during or subsequent to the development of the dysenteric process. Some look upon these secondary processes in relation to the dysentery as in the relation of effect following a cause; or that there is an immediate and direct connection between the primary dysenteric process and the secondary lesion. Such a relationship has not been shown to exist in all cases; and it is more probable that the *dysenteric* process, when it operates on the system during a protracted period, predisposes, as many other morbid states do, to the development of secondary local lesions in distant parts.



from the morbid intestines. But the evidence tends rather to show that the hepatic lesion and the dysentery are each excited by the same cause. If Dr. Budd's theory were correct, we ought to have liver abscess a common occurrence after ulcerations of typhoid fever, and after those of tuberculous lesions of the intestines; but we do not find that such a lesion of the liver is usual in such cases.

Regarding hepatic complication in dysentery, the following conclusions may be stated: (1.) That dysentery, in a great number of cases, more than half, commences and runs its course complicated by obvious functional hepatic disease; (2.) That the hepatic disorders and the dysentery acknowledge a common cause and disease-process; (3.) That about 18 per cent. of the fatal cases of *dysentery* are complicated with hepatic abscess; and about 57 per cent. with hepatic lesions; (4.) That in a few of these cases ulceration of the intestine may be the primary disease, and the source of the hepatic abscess by the phenomena of thrombosis and embolism in connection with the pelvic veins and veins of the mesocolon.

The occurrence of hepatic abscess with dysentery has been generally viewed as a result of phlebitis; but Dr. Parkes, after the most careful observation of such cases, never found the slightest trace of inflammation in the small veins of the intestines, while no direct proof has been advanced of the mediation of the portal blood in the process; and in conclusion, writes Dr. Hensch, "I believe we must give the preference to that view which regards the two diseased processes, dysentery and abscess of the liver, as running their course together, dependent upon one and the same cause; in favor of which view is the circumstance, that in hot climates abscess of the liver very frequently occurs associated with remittent fevers, or consecutive to them, without dissection exhibiting any ulceration of the mucous membrane of the intestines" (*Brit. and For. Med.-Chir. Review*, July, 1854). The comparative frequency of the occurrence of hepatic abscesses may be seen from the following statement:

In *Calcutta* General Hospital they occur at the rate of 13.1 per cent. (MACPHERSON); in the Medical College Hospital, at the rate of 25.9 per cent.; in *Bombay* General Hospital, at the rate of 40 per cent. (MOREHEAD); and in *Madras* Presidency, at the rate of 50.97 (ANNESLEY), 19.35 (PARKES), 17.9 (INNES, at Secunderabad); MACNAMARA, in Madras, 50.9 per cent.; French surgeons in Algeria, 12.7 per cent.; EYRE, of the Madras Fusileers, 22.8; WARRING, in various localities not stated, 23.5 per cent.; STOVELL, in European General Hospital, Bombay, 19.3 per cent.; LEITH, in Bombay, 8.5 and 15.2 per cent.; and MARSHALL, in Ceylon, 28.8 per cent.

Too much attention and importance seem to have been put upon abscess of the liver *per se*, irrespective of other obviously morbid conditions of that organ—*e. g.*, impaired functions, congestion, enlargement. To regard secondary hepatic abscess as due to absorption of pus or other morbid matter from ulcerating mucous membrane, or to a true phlebitis, is to take too narrow a view of the relation of liver disease to dysentery; for if we are to judge by the condition of the bile alone, the liver is diseased (in function, at



Dysentery "is found to complicate readily in all climates with the prevailing fevers." Within the tropics it is frequently associated with *remittent* and *intermittent fevers*; in the geographical region of *typhus fever* it is a most frequent complication, under various circumstances, and becomes capable of propagation from person to person; and, lastly, it is also occasionally complicated with *scurvy*. When dysentery follows upon, or is associated with, intermittent fever, the spleen will frequently become enlarged, indicated in the outset by general anæmia, or splenic cachexia, with a low asthenic type of dysentery.

The scorbutic complication is developed in cases of *dysentery* when the supply of food has been deficient in fresh vegetables, or when it consists in whole or in the greater part of salted meat. Sir Gilbert Blane asserts that the complication has been known to arise among prisoners of war, living entirely on fresh (animal) diet.

"The most terrible instance of suffering from this cause," writes Sir Ranald Martin, "was that of the European portion of the force employed in Ava during the first Burmese war, where they were for six and a half months fed on salt rations, and where 48 per cent. perished within ten months, principally by dysentery with the scorbutic state." Such disasters have since been equalled, if not surpassed, by the sufferings of our troops in the camp before Sebastopol during the winters of 1854-55, under the influence of exposure, fatigue, and continued rations of salt meat and green coffee.

There is still another light in which the pathology of this disease requires to be studied, namely, in the

**Types and Forms of Dysentery.**—These have been variously described as (1), *the purely inflammatory, acute, hyperacute, or sthenic form*. In this form, while the phenomena indicate acute and severe inflammatory action, there is no tendency to the great depression of the nervous, circulatory, and muscular functions, which gives a marked character to some of the other types of the disease, such as (2), *the asthenic forms*. In the *asthenic* forms, besides the depression of the functions just noticed, there is much greater tendency in these forms to spread by infection, or under an epidemic influence. These asthenic forms are sometimes described as *adynamic, typhoid, malignant, bilious, intermittent, or remittent*, according as certain phenomena prevail characteristic of these states.

**Symptoms of Dysentery.**—An ordinary attack generally commences with *diarrhœa*; but in twelve or twenty-four hours disagreeable feelings begin to attend the frequent loose discharges from the bowels. These are irregular pains, commonly called "gripes," along the course of the large intestine, and sometimes described as "shooting," or "cutting." Technically, such symptoms are known as *tormina*. They are momentarily relieved by discharges from the bowels. But after a short time a sense of heat ascends from the rectum, and pain extends to the epigastrium, till the whole abdomen is painful. There is a frequently returning inclination to go to stool: the griping and straining continue without the patient being able to pass anything more than a little bloody mucus. These symptoms, are generally aggravated during the night and early



Clouston some of the patients had ordinary diarrhœa (diarrhœa of irritation?), from periods varying from two or three hours up to twenty-four hours, before blood appeared in the stools. In some cases there was great pain in the abdomen for twenty-four hours before the diarrhœa set in (evidence of irritation?). In other cases there was scarcely any pain at any period of the disease.

Dr. Clouston distinguished two classes of cases. "In the first the patient had two or three loose stools, or perhaps had no ordinary stools at all, but at once began to pass glairy mucus, mixed with blood, in small quantities at a time, from the bowel. He had no pain, no fever, no want of appetite, and he refused to believe he was ill. This would continue for a day or two, and then the blood would increase in quantity, and the stools would become more frequent. Pain would begin to be felt in the region of the rectum, and the pulse would mount up by ten or twelve beats. For days the patient would be at stool every hour or two, and of course would become weaker. His tongue was then seen to be coated with a dirty yellowish-white fur; but the appetite for such forms of nourishment as milk, strong beef tea, calves' foot jelly made with wine, was still good. Solid food was not relished. These stools would then be seen to be coated with a semi-fibrinous semi-purulent-looking membrane. The tongue would then become clean, and glazed, and beefsteaky; the evacuations became feculent, mixed with pus, the latter element becoming gradually less as the patient advanced in his slow convalescence.

"In the second class of cases the patient had from the first great pain in the abdomen, of a griping kind, a hot skin, and a pulse over 100; the dejections were copious, and frequent, and watery, while they were largely mixed with blood. In many cases there was sickness; in all, loss of appetite. After some days the tongue and mouth would become dry, and parched, and black; the features pinched; the pulse small and quick; and death soon ensued. In some cases the stools would, after a time, become membranous and shreddy, and then purulent, till the patient was more gradually weakened and exhausted. One such case lived six weeks, another two months. In one only of this class of cases (out of seventeen) did the patient recover.

"All the cases had the following features in common: Bloody stools at first, tending to become purulent; intense fetor of the evacuations during the whole of the disease; no scybala, and great thirst" (*Med. Times and Gazette*, June 10, 1865). These were cases of dysentery caused by the poison of animal effluvia from decomposing human excreta (undiluted) acting on constitutions in which vegetable diet seems to have been deficient (scorbutic), and in whom the nervous power was below par.

If the disease proves fatal in the chronic form, the patient generally becomes rapidly altered and prostrated by his sufferings, is strikingly emaciated, and often earnestly prays to be relieved from a life disgusting to himself and entirely despaired of by others. Death begins at the heart.

The patient, on the other hand, may in a few rare instances re-





vice in the field, have at all times been found to be conditions powerfully predisposing to dysentery.

The effects of *salt* diet in the production of dysentery being less known than the other predisposing causes, it may be as well to state that, by an experience of twenty years in the West Indies, it has been determined that in the Windward and Leeward Command, when the rations issued to the troops consisted of salt provisions five days in the week, the mortality from diseases of the stomach and bowels among the officers was as two to four per cent., while that among the soldiers was as 20.7, or a tenfold ratio. On the contrary, in Jamaica, when salt provisions were issued to the troops only two days in the week, the mortality from the same diseases approximated so nearly between these two ranks as to be almost an equality. And corresponding facts to these have been observed in Gibraltar, on the coast of Africa, and at St. Helena. The Sierra Leone Commissioners on the western coast of Africa, who investigated this subject on the spot, were of opinion that the large proportion of salt rations mainly contributed to the sickness and mortality from diseases of the stomach and bowels in the form of dysentery and diarrhœa; and the following statement, given by the late Sir Alexander Tulloch in his Statistical Reports (page 11) on the sanitary condition of the troops in the West Command, shows the marked reduction which took place in the deaths from this class of diseases subsequent to the introduction of fresh meat diet; the mortality being reduced to a *tenth* part of its former ratio:

PREVIOUS TO ALTERATIONS IN RATIONS.						SUBSEQUENT TO ALTERATIONS IN RATIONS.										
Year.	Mean Strength.	Dysentery and Diarrhœa chiefly.		Ratio per 1000 of Mean Strength.		Year.	Mean Strength.	Dysentery and Diarrhœa chiefly.		Ratio per 1000 of Mean Strength.						
		Admitted.	Died.	Admitted.	Died.			Admitted.	Died.	Admitted.	Died.					
1825	571	235	32	411	56	1828	232	189	1	600	5.16					
1826	471	256	26	543	56	1829	114	50	—	439						
1827	845	209	18	606	38	1830	42	22	1	524						
Total,	1387	700	71	Average. 505	Aver. 51	to 1836										
						Total,	388	211	2	Average. 548	Aver. 5.16					

In the navy the same effects of ill-regulated diet have been observed, and the good effects of a change. “In 1797,” says Dr. Wilson, “the victualling (of the navy) was changed, greatly improved, and consequently immediate to the change the health of the seamen improved strikingly. *Scurvy, typhoid fever, dysentery, and ulcer*, which, up to the period of the change, had produced great havoc, became comparatively rare in occurrence and light in impression,” and, it may now be added, are hardly known except by name.\*

\* As Dr. Christian justly observes, the salt meat of military and naval rations is not the same as the salt meat of civil life. The former is highly salted, in order to



Drained from swamps, and used for drinking and cooking purposes, as it was on the Chinese coasts, it exerted a marked injurious influence both in exciting and in maintaining the disease. In connection with impure water, the reader is requested to refer to what is said afterwards under the head of "Parasitic Diseases;" and especially under "*Distoma hæmatobium*."

Many other predisposing causes favor the development or propagation of the disease, especially amongst soldiers in active service—namely, long marches in hot weather, bivouacking at night in the open air (often extremely cold both absolutely and relatively to the day), want of sufficient clothes and bedding, may be mentioned as the chief.

It does not seem to be so clearly understood as it ought to be, that dysentery is contagious, or rather that it is capable of being propagated from person to person. Being a frequent complication or concomitant of contagious fevers, it has been believed to inherit similar contagious properties. In the severe form of dysentery, for which the old Infantry Barracks of Secunderabad, in the Deccan, have long been notorious, it has been observed that men, laboring under other diseases, who happened to be exposed to the putrid effluvia of the excretions of dysenteric patients, were often severely affected by the disease (W. C. MACLEAN). There is, therefore, good reason to believe that the exuviae of dysenteric patients, as passed by stool, may, like those of typhoid fever, propagate the disease; and the observations of Budd and Goodeve give support to this view.

**Prognosis.**—The prognosis in dysentery depends much on the country in which the disease occurs, and on the combination of circumstances predisposing to the disease, not less than on the form or type which the disease may assume. In hot climates it is calculated that the deaths vary from one in nine to one in twenty; and on actual service the chances of recovery are much diminished. In all returns, however, the total deaths recorded give a faint idea and inaccurate representation of the real mortality resulting from dysentery. If it were possible to trace out the men who were invalided from the army and navy services from the effects of this disease, it would be found that the mortality is very much greater than is represented by tabular returns. It is a malady which, once fairly engrafted on the system, never leaves it till life itself becomes extinct (BRYSON, and others). It is sometimes also insidious in its mode of attack and progress; and there is such a desire, on the part of soldiers especially, to avoid the restraints of hospitals, that the disease is sometimes beyond the power of medicine before coming under treatment, especially in tropical commands (TULLOCH). In the cases described by Dr. Clouston as those of the second class (see page 557, *ante*), the chances of recovery are hopeless from the first, and all complications with scorbutus are very unfavorable. There may be diseases of a more rapidly fatal character, but there are few which entail so great an amount of suffering. When once the disease has passed into the chronic form, it slowly, but not the less surely, continues, by a most loathsome process, to exhaust the vital energies, until death relieves the patient of an existence rendered almost in-



“In acute cases,” says Sir James McGrigor, “we commenced by copious venesection, and immediately afterwards gave twelve grains of compound ipecacuanha powder every hour, which was repeated three times, with plenty of barley-water, and profuse sweating was encouraged for six or eight hours. A pill of three grains of calomel and one of opium was administered every second night, and in the intervening day, two drachms of sulphate of magnesia dissolved in a quart of light broth. The venesection was repeated, while the strength and pulse permitted it, until the stools were free, or nearly free, from blood. Dover’s powder as a sudorific was always given after bloodletting.

“In cases where the pains were excruciating, and attended with tenesmus, the warm bath gave instantaneous relief. This plan being steadily persevered in for a few days, the inflammatory diathesis of the intestinal canal, which had excited symptomatic fever throughout the general system, was found gradually to be relieved, and paved the way for returning health. In this stage gentle tonics, with light nourishing diet cautiously taken, and at first given in moderate proportions, were administered with the happiest effects.

“The disease was not unfrequently cut short by this method. If, however, the disease became chronic, a different mode of treatment was pursued, and not unsuccessfully, if the disease had not been of long duration, the intestinal canal not much disorganized, or not complicated with other diseases.

“The first indication in this chronic state was to relieve the tenesmus and procure easy stools, and with this view ipecacuanha was given, sometimes with calomel, sometimes without it. The neutral salts were given, or castor oil, jalap, and various other medicines of the same class. The second indication was to relieve the number of the stools, and to restore tone to the alimentary canal. With this view Dover’s powder, the compound powder of chalk with opium, astringents, and demulcents, with aromatics, were given, occasionally interspersing laxatives, and obviating particular symptoms as they occurred. Lastly, an infusion of bitters was given, to restore tone to the relaxed intestine.”

In addition to these remedies, Sir James McGrigor states that the *balsam of copaiba*, an *infusion of calumba*, *hæmatoxylon*, *kino*, and *catechu*, assisted by *opium* occasionally, gave much relief, and also the administration of a variety of enemata, and especially one of a strong solution of *superacetate of lead*; while in cases of liver affection, he adds “that friction of the abdomen with mercurial ointment gave the least irritation, and at the same time produced less debility.”

Such is the statement of the practice pursued in dysentery during the Peninsular war, on a scale whose magnitude has seldom been surpassed even in modern times. If, however, we look to the returns, we find it highly probable that not more than two out of three of those attacked ultimately recovered.

The bark of the root of *calotropis gigantea* (or *mular*) has been recently used in India, and found to be an excellent substitute for *ipecacuanha*. It is used in doses of a scruple to a drachm, is a relia-



## [CHRONIC CAMP DYSENTERY.]

(DR. CLYMER.)

**History.**—The extreme frequency and mortality of this disorder in the armies during the late civil war give it interest and importance to the American physician. More than one-fourth of all the cases of disease

---

*the most sthenic forms of the disease, and confine the use of leeches within the narrowest limits.*

**Mercury.**—It is certain, too, that mercury is yearly less and less used in India than it was, and there is much evidence to show that a corresponding reduction in the mortality of the disease has been the result. The objections to its use are numerous,—it entails great suffering on the patient, if pushed to ptyalism, aggravating his miseries, and too often permanently injuring his constitution; it has no specific action on the disease, and its cholagogue effects can be attained by remedies which are not open to such objections as can be brought against mercury. (With regard to its cholagogue effects, grave doubts are thrown by the experiments of Dr. George Scott, referred to at the footnote of page 160 by the author.) In sloughing dysentery it is followed by the worst results; and the observations of clinical observers in India have shown that individuals under the influence of mercury are not only not exempt from attacks of the disease, but are peculiarly prone to be affected by it. This is the case in a very marked degree in Asiatics (MOREHEAD and MACLEAN).

**Ipecacuanha.**—This remedy has long been used in South America in the cure of dysentery,—whence, indeed, it came. It was much used in India until the mercurial notions of James Johnston prevailed. It was again used by Dr. Twining, of Bengal, by whom it was strongly recommended, and also by Dr. Mortimer, of Madras. Twining combined it with blue pill and gentian, and used it chiefly in small and oft-repeated doses. In South America the practice has always been to administer an infusion of the bruised root,— $\mathfrak{z}\text{ij}$  being infused over night in  $\mathfrak{z}\text{iv}$  of water, and given early in the morning. In Peru it is given in doses of  $\mathfrak{z}\text{ss.}$  to  $\mathfrak{z}\text{j}$  of the powdered root in a little syrup and water. This practice of giving ipecacuanha in large doses has lately been revived in India with encouraging success, and, I believe, the greatest number of cures. It appears to act on the portal capillaries, and on those of the mucous membrane of the bowels, and to determine powerfully to the skin. *It is usually given in doses of half a drachm or a drachm, either in pills or bolus, or suspended in mucilage, according to the fancy of the patient. It is advisable to give an opiate half an hour before, and to withhold all drink for some hours.* Unless there be hepatic complication, it seldom happens that much vomiting is caused by these large doses; on the contrary, they are often tolerated when smaller doses are rejected. The dose should be repeated in about six hours. A sufficient interval should be allowed to intervene between the doses of ipecacuanha to admit of the patient being sustained by nourishment adapted to the stage of the disease. I need hardly add a caution not to press the remedy too far.

“Dr. Cornish, of the Madras army, has shown from official documents that the mortality from acute dysentery in Southern India under mercurial treatment was 7.1 per cent. Since the general introduction of ipecacuanha in full doses it has fallen to 1.8. Dr. Ewart, of Bengal, has shown that equally good results have followed the same system in that Presidency. During the forty-two years from 1812 to 1853-54 the mortality among European troops in the Bengal Presidency amounted to 88.2 in the thousand. But during 1860, when large doses of ipecacuanha were administered, the mortality was only 28.87 in the thousand.

“Great credit is due to Mr. Docker, of the 7th Royal Fusiliers, for recalling practitioners to the use of this invaluable remedy (*vide Grant's Annals of Military and Naval Surgery*, vol. i).

“In no disease is early treatment more necessary than in dysentery, and I believe that, if conducted as above, except in the malignant and ‘putrid’ forms, we may look for good results in a large proportion of cases. Turpentine epithems and fomentations should be diligently used, and the patient's strength should be supported by nourishment of a bland kind, suited, in degrees of nutritive value, to the stage of the disease.

“In the scorbutic form we have a valuable remedy in the *Bael fruit*, when procurable. This fruit contains a large quantity of *tannin*, with vegetable mucus, a bitter principle, and a vegetable acid. It is much used in Bengal; and in the





meat and vegetables were deficient, but also by the presence of well-marked scorbutic phenomena. The influence of climate and season is abundantly proved by the detailed statistics of individual armies and localities. The amount of greatest prevalence in any given army was also the month of the greatest prevalence of intermittent fever.

Like camp fever and intermittents, diarrhœa and dysentery were most frequent in the Central region, less so in the Atlantic, and least in the Pacific region. In the Central region the cases were more numerous than the strength during the first year, and nearly equal to the strength during the second; in the Atlantic they were more than half the strength during the first year, and more than three-quarters during the second; in the Pacific region they were each year somewhat over one-quarter the strength. The differences between the ratio of mortality to strength in the three regions was still more striking. In the Central region there was 9 per 1000 of mean strength during the first year, and 23 per 1000 the second; in the Atlantic 1 per 1000 during the first year, and 9 per 1000 the second; in the Pacific region less than 1 per 1000 during each year. The ratio of deaths to cases shows strikingly the influence of region.

RELATION BETWEEN CASES AND DEATHS OF DIARRHŒA AND DYSENTERY.

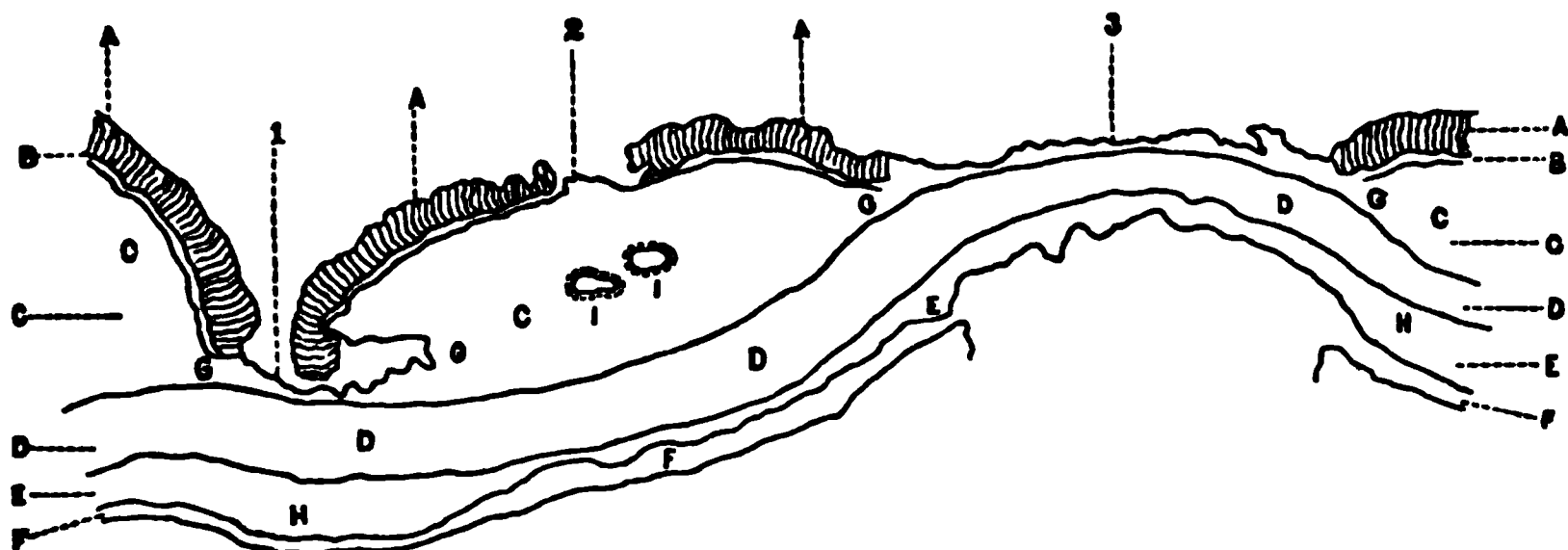
	YEAR ENDING JUNE 30, 1862.		YEAR ENDING JUNE 30, 1863.		ANNUAL AVERAGE FOR THE TWO YEARS.	
	Ratio of deaths per 1000 cases.	Number of cases to each death.	Ratio of deaths per 1000 cases.	Number of cases to each death.	Ratio of deaths per 1000 cases.	Number of cases to each death.
Atlantic region, .	2.07	483.09	11.84	84.46	8.60	116.28
Central region, .	9.66	103.52	27.54	36.31	22.85	43.77
Pacific region, . .	2.75	363.64	3.00	333.33	2.89	346.02
Total, . . . .	5.55	180.18	20.31	49.24	15.93	62.77

The influence of season in each of the regions was most marked. Dysentery and diarrhœa were by far most frequent in the summer and autumnal months. In the Atlantic region the greatest monthly ratio was during July, 1861, after which it diminished through the fall and winter, but again increased from March to June, 1862; July, 1862, was the maximum month for the second year. The cases greatly diminished in August, after the Army of the Potomac had withdrawn from the Peninsula to near Washington; but increased in September, and attained a second maximum in October, which is the month in which intermittents were most frequent in this region; it then diminished steadily till April, 1863, increasing again in May and June. In the Central region the maximum month is August, 1861, after which there is a gradual diminution till December, an increase in January, 1862, a falling off in February, and a great increase in March and April. The monthly ratio became gradually less during May, June, July, and August, but increased considerably in September, after which it gradually diminished until January, 1863, when it increased again, and made subsequently but slight fluctuations. On the Pacific coast the disease was most frequent during the summer and autumnal months. The following tables give the rates:



generally of a slate, dark-red, brownish, or greenish-brown color; the base of the ulcers is yellowish or yellowish-brown, often with brown or blackish sloughs adhering to their surface or edges. This group represents a more advanced stage of the disease. (3.) In addition to the lesions of the first and second groups, the surface of the large intestine is more or less coated with a yellowish, or greenish-yellow, or brownish-yellow croupous, pseudo-plastic, caco-plastic, or false membrane, similar to the membrane formed in the air-passages in diphtheria, and which sometimes extends to the small intestines, and is generally found in those who have died during the supervention of acute dysenteric symptoms. Examination by the microscope of properly prepared sections shows it to be composed of innumerable round cells (lymph-cells, pus-cells), held together by an adhesive granular matrix, more or less re-

FIG. C.

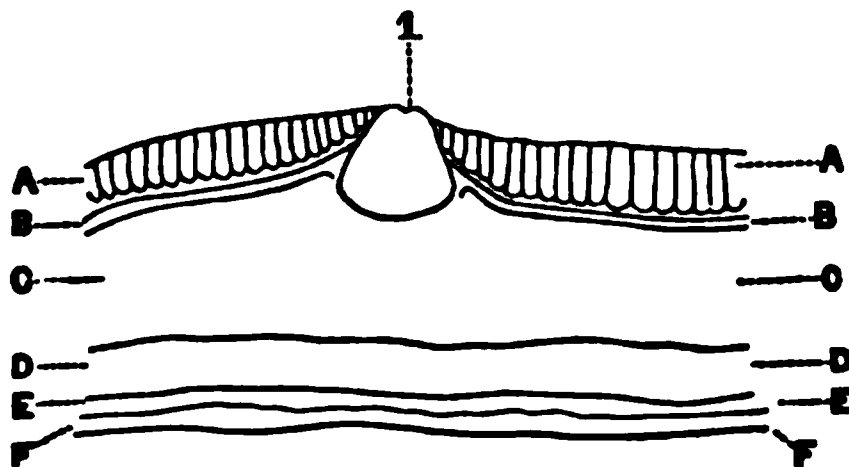


Perpendicular section of ulcerated colon in Chronic Camp Diarrhoea, cut transversely. A A A A. Follicles of Lieberkühn. B B. Muscle of Brücke. C C C C. Submucous connective tissue. D D D D. Circular muscle of the colon. E E E. Longitudinal muscles, cut transversely; at H H, two of the longitudinal bands of this layer. F F F. Peritoneal layer. G G G G. Points of very active cell multiplication. I I. An artery and vein cut through transversely. 1. A deep follicular ulcer, extending to the muscular layer. 2. A small superficial ulcer, which has penetrated to the submucous connective tissue. 3. An ulcer of great size, penetrating nearly to the muscular layer, which is covered with granulations. (Woodward.)

sembling coagulated fibrine. The origin of this membrane may be traced to a rapid multiplication of epithelial cells and superficial connective tissue corpuscles of the diseased mucous membrane.

Two forms of ulceration are observed in the colon: in the first, the process begins in the closed follicles; in the second, in the intestinal epithelium or the glandular layer. The closed follicles enlarge by multiplication of their cellular elements till they project as little tumors above the surface, as shown in Fig. D, in which is the enlarged solitary follicle. The tumor, having enlarged to a certain extent, ruptures; its cellular elements escape, and a minute ulcer is formed, as shown in Fig. E, in which is the ruptured follicle, with the minute ulcer. The cells or corpuscles of the connective tissue surrounding the enlarged follicle now multiply, and the ulcer spreads by the superficial cells floating off into the intestinal cavity, while a new base is continually formed by the multipli-

FIG. D. (Woodward.)





(nitric and sulphurous), the nitrate of silver, the oil of erigeron canadensis, persulphate and perntrate of iron, &c. When present, as they generally are, the malarious and scorbutic phenomena must be specially treated. Climate is the essential element in the treatment; its influence is absolute and abiding; and without it permanent results are rarely, if ever, attained. The value of climate in chronic camp dysentery was recognized during the Mexican war; and during the late civil war, the Medical Department of the Army was fully alive to the advantages to be derived from it; and it is conclusively shown that the number of recoveries bore a direct relation to the latitude of the climate, and its freedom from paludal poison, in which the patients were treated. Soldiers with chronic diarrhœa were, in the Atlantic region, transferred to the hospitals in the State of Vermont, and in the Central region, to high northern regions, Keokuk, Ia., Madison, Wis., Detroit, &c., and with favorable results.

During the second year of the war, the proportion of deaths from chronic dysentery in the general hospitals of New England, was 1 to every 48.8; New York City and State, and New Jersey, 1 in 18.7; Pennsylvania and Delaware, 1 in 15; Maryland and District of Columbia, 1 in 11.4; the Virginia, North and South Carolina coast, 1 in 7.1. The writer's experience during two years as Medical Director, Department of the South, was that under no medical or dietetic treatment was a single case cured, and but few benefited; the mortality being very great, until the plan of sending all the cases of the disorder, as soon as it became developed, to Northern hospitals. In the Central region the relative influence of latitude was observed, though the mortality was greater, being 1 in 8.53 in the hospitals of Cincinnati, Ohio; and 1 in 4.7 in those of New Orleans, Louisiana. In the hospitals of Keokuk, Ia., the death-rate was 1 in 9.1; in St. Louis, 1 in 5.2; and in Cairo, situated on the alluvial peninsula formed by the junction of the Mississippi and Ohio rivers, and where the most intense malarial influences prevail, it was 1 in 3.98. The diet should be antiscorbutic, and fresh vegetables and ripe fruits are useful; fresh meat, well minced, and broth made from it and not from any extractum carnis, with milk, cream, and eggs, should be allowed, according to the state of the digestive organs. The sulphites in weak vegetable bitter infusions, will check the fermentative and putrefactive tendency in the stomach and upper intestines, and aid digestion.]

### DIARRHŒA.

**LATIN**, *Alvus soluta*; **FRENCH**, *Diarrhée*; **GERMAN**, *Durchfall*—Syn., *Diarrhœe*; **ITALIAN**, *Diarrea*.

**Definition.**—*A frequent discharge of loose or fluid alvine evacuations, without tormina or tenesmus.*

**Pathology.**—This affection is rather a consequence or a symptom of certain pathological states than of itself a disease; yet, as there are many agents, both of a moral and physical nature, that act thus upon the human body; and as there are also many known morbid poisons which bring about this state, it merits some notice in the class of diseases now under consideration. It is a morbid action of function, rather than any disease of structure, being unassociated with any definite specific lesion of vital parts. It may be regarded



variety. It corresponds to the "*diarrhœa lienterica*" of the older authors. The most marked and characteristic phenomena which attend the disease are due to the almost total suspension of the digestive, assimilative, and absorbent functions, the *egesta* often differing but little in appearance from the *ingesta*. Such a form of diarrhœa occurs more frequently in children before the period of the second dentition than at later periods. It is frequently the consequence of previous inflammatory irritation of the alimentary mucous surface, and disease of the mesenteric glands. It seems as if, in this variety, the stomach had lost its tone or vital energy, as well as the mucous membrane of the alimentary canal; and it no doubt results, in the first instance, from indigestion. This was a frequent form of diarrhœa amongst the soldiers in the Crimea, as observed by Dr. Lyons; and the soldiers themselves observed it, and were in the habit of saying, "It is of no use eating, as our food passes through us in the same state as it goes in." The appetite is usually voracious; and when this form of diarrhœa continues long, the debility becomes extreme; and when death takes place, it is from stupor and exhaustion.

In a practical point of view, these are the principal varieties of idiopathic diarrhœa which require to be distinguished; and the diagnosis of the form of diarrhœa symptomatic of the invasion of other diseases are noticed under the special diseases of which they form a part.

**Treatment.**—For practical purposes, the treatment of these three forms of idiopathic diarrhœa may be founded on the following indications—namely, *first*, that in which the *tongue is clean*, the pulse quiet, and all constitutional reaction absent; and, *second*, that in which the *tongue is white* and coated, the pulse accelerated, some fever present, and the pain or soreness constant and increased by pressure. The stools in either case may be black, green, white, or mixed with blood indifferently.

When the *tongue is clean*, if the disease be quite incipient, the usual practice is to give one dose, consisting of an opiate, combined with a gentle cathartic. The form may be *one grain of opium*, combined with a *drachm of compound rhubarb powder*, or combined with *five grains of calomel*. To remove any offending matter that may be present, their action may be aided by castor oil, or a saline cathartic, such as a Seidlitz powder. Sometimes it may be advisable to omit the opium, and to combine antacid remedies with the purgative, as in the following prescriptions:

R. Sodæ Bicarbonatis, Hydrargyri cum cretâ, ãã gr. ij—ad gr. v;  
Magnesiæ Carbonatis, gr. iij—ad gr. vj; Pulv. Rhei, gr. v—ad gr. viij;  
*misce.*

Or:

R. Sodæ Bicarbonatis; Pulv. Rhei; Pulv. Calumbæ, ãã gr. iv—ad gr. vi; *misce.*

The administration of such a powder may be repeated at intervals—twice or thrice a day; and ipecacuanha in small doses (a





Sulphuric acid, in doses of the officinal diluted drug, of twenty to thirty drops, with water simply, or combined with the compound tincture of gentian, has been found a useful remedy. The sulphuric acid may be alternated with the nitro-muriatic acid, and prescribed in a similar manner.

The dietetic treatment should be limited to slops, puddings, and whitefish boiled, and the drink to weak brandy and water, which acts locally as an astringent, and generally as a diffusible stimulus.

### MALIGNANT CHOLERA.

**LATIN.** *Cholera pestifera*—Idem valent, *Cholera serosa*, *Cholera spastica*, *Cholera asiatica*; **FRENCH,** *Choléra asiatique*; **GERMAN,** *Cholera nostras*—Syn., *Cholera*, *Asiatische Cholera*; **ITALIAN,** *Colèra morbus*.

**Definition.**—A disease essentially of miasmatic origin, developed under certain atmospheric and terrestrial local conditions in Europe, Asia, and America, and capable of being propagated or diffused, to a certain extent, over the surface of the earth, through the atmosphere, or in some other way, and also by means of human intercourse between the healthy and the sick. It is characterized in many (but not in all) cases by premonitory diarrhœa, sudden muscular debility, tremors, vertigo, occasional nausea, and spasmodic pains in the bowels, depression of the functions of respiration and circulation, and a sense of faintness; copious purging of serous fluid and sometimes blood, succeeded by vomiting and burning heat at the stomach, coldness and dampness of the whole surface of the body, coldness and lividness of the lips and tongue, cold breath, a craving thirst, a feeble rapid pulse, difficult and oppressed respiration, with extreme restlessness (a state expressed in physiological language by the term “anxietas”), suppressed urinary secretion, blueness of the entire surface of the body, a sunken and appalling countenance, a peculiarly suppressed voice, a peculiar odor from the body, partial heats of the præcordia and forehead—fatal collapse, or reaction and secondary fever. Under conditions favorable to its development it often becomes epidemic.

**Pathology.**—During recent years volumes have been written on the nature and causes of cholera; and the description of it which I here give is chiefly drawn from the writings of men who, while they have been themselves original observers of the disease throughout an extensive experience in this country, in India, and in the north of Europe, have since been the able expositors and philosophical critics of the numerous official, original, and independent scientific accounts of it which have been lately given to the world.

The remote cause of cholera is unquestionably a poison; and that it should spread over countries which, in respect to climate, soil, geological formation, and also as to the moral and physical habits of the population, are the most opposite to those where it first originated, is only explicable by the hypothesis of its propagation by a specific poison.

One doctrine, therefore, now very generally accepted regarding



term "*algide*," first used by the French pathologists, very properly designates one of its most remarkable and constant symptoms—namely, the diminution of animal heat. The sensation of cold communicated to the observer has been compared to that experienced on touching a moist bladder or the skin of a frog. The algide symptoms, therefore, essentially constitute the characteristic phenomena of this disease. In proportion to them is the malignity and rapidity of the case. They afford the only measure of its severity, and from them only can a correct prognosis be formed. The vomiting, purging, and cramps are considered as non-essential phenomena; for authentic cases of cholera are on record entirely divested of these symptoms; and the suddenness with which the poison sometimes extinguishes life is extremely remarkable. Instances of death taking place in two, three, four, or six hours, are by no means uncommon. When the disease broke out at Teheran, in May, 1846, Dr. Milroy states that those who were attacked dropped suddenly down in a state of lethargy, and at the end of two or three hours expired, without any convulsions or vomitings, but from a complete stagnation of the blood. In Bulgaria, during the outbreak of cholera in the allied armies, in the summer of 1854, the rapidly fatal character of the early cases was notorious. Such records confirm the views developed by Drs. Parkes, Johnson, Sir Thomas Watson, and others, as to the essentially poisonous nature of the disease, and the very rapid depressing influence of the poison. It is plain, also, that a poison so powerful, so suddenly overwhelming all Nature's efforts at resistance, does not allow time, in many cases, for any secondary or specific actions to be set up.

After death, during collapse, the following lesions have been noticed: The follicular structure of the intestinal canal has been found to be swollen, and the intestine filled more or less with a turbid, inodorous, semi-diaphanous fluid, usually compared to a thin starch or rice-water, the remains of that immense secretion which has taken place during life, and which, being tested, has been found sometimes acid and sometimes alkaline. It is found in its most unmixed condition in the small intestines. It consists of a thicker and thinner portion, and it appears to be the latter which chiefly constitutes the "rice-water" stools, which may be passed off without admixture of the thicker substance. A layer of grayish mucus has also been found coating the whole of the mucous membrane of the alimentary canal, but without a trace of bile, although the gall-bladder is usually filled with that fluid. If the first stage has been prolonged, the mucous membrane of the alimentary canal is of a livid color, and in some instances has presented a mammillated appearance, caused by an enlargement of the tubular glands, from which a white opaque fluid can be squeezed out and the mammillated appearance effaced.

The liver, the spleen and the kidneys have in general been found gorged with blood as to their veins, and the veins of the kidneys are quite as visible as from "contracted mitral-valve" disease (SUTTON); and this engorgement extends even to the bones, which, Louis says, appear as if the animal had been fed on madder. The capillary



of collapse in cholera, from an original drawing by Mr. John Wood. The lungs weighed very much less than usual, often not half their usual weight; and on section they appear dry. The blood they contain is all in the branches of the pulmonary arteries: it is black-looking blood—thicker than usual, but still fluid, so that on puncturing a vein, such as the jugular, it escapes in such quantities that the right side of the heart is emptied in a few minutes (SUTTON). The anterior portions of the lungs were of a gray color—very much paler than normal: the posterior portions and the bases of the lungs were much darker in color, soft in consistence, and easily broken down. A section of the lung substance rapidly became of a bright scarlet color on exposure to the air.

Such are the appearances which the body has presented when the patient has died in the first, the asphyxiated, pulseless, or collapse stage. The enlargement of the solitary follicles of the intestines is believed to be peculiar to those cases in which diarrhoea, or other disorder of the alimentary canal, had for some time preceded the fatal attack. This enlargement bears no relation to the intensity of the disease, being often most conspicuous in the least severe cases; and it is an appearance now considered of secondary importance, and consequent on the purging. In the experience of Dr. W. T. Gairdner it has been found in about two-thirds of the cases.

When the patient has survived until reaction has taken place, and the second or febrile stage has been formed, the body no longer presents that shrunk, worn, and livid appearance it did on death taking place in the first stage; but, on the contrary, rather a full and plump appearance. The injection of all the large organs disappears, the blood being recalled to the surface of the body. The alimentary canal is no longer distended with the turbid secretion peculiar to cholera, but contains a thin yellowish *purée* of fecal matter, having the usual odor. The mucous membrane of the alimentary canal has now, however, been found more or less diffusely inflamed, sometimes in all its divisions, but more especially in the pyloric portion of the stomach, and also in the duodenum. The glands of Peyer, as well as the solitary glands, though occasionally found enlarged, were seldom found ulcerated; but when that was the case the corresponding mesenteric glands were also enlarged, being sometimes pale or purple, and when cut into, gave issue to a dark liquid blood.

The post-mortem appearances, and the order of the symptoms tend to show that the blood is obstructed in its passage through the lungs; and that the loss of animal heat, embarrassment of the respiration, and gradual arrest of circulation, are produced by some aberration of the proper respiratory changes, or impediment to them. But as the mechanical part of respiration remains perfect, and as there is no impairment in the voluntary command of the respiratory muscles, and as the heart evidently beats in many cases till stopped by the want of blood on the left side, and by its accumulation on the right side, "we are compelled to look," says Dr. Parkes, "for the cause of such arrest of the circulation in the only remaining element of respiration—namely, in the blood itself"



served by Dr. Parkes as occurring in little less than a quarter of the whole number of cases observed by him. The presence of fibrine in the blood was not indicated by any coagulation either in or out of the body; and, whether coagulated or not, the blood has usually a dark color; but it generally acquired an arterial tint when brought into contact with the air in thin layers. Dr. Robertson's observations were made on the cases occurring in the Edinburgh epidemic of 1848 and 1849; while Dr. Parkes's observations were made on two severe epidemics of cholera in India in 1843 and 1845. He also made the interesting observation, that a few drops of the thick substance taken from the intestines had sometimes the effect of restoring the vivid arterial color of the blood. During the transudation into the intestinal canal, it appears that the diffusion currents *from* the blood into various structures are diminished; while, on account of the density of the blood, the inverse currents *from these structures to* the blood are augmented in rapidity. In this way fluids are drawn from the muscles, the viscera, and, in fact, most of the tissues; and it is probable that these fluids are charged with substances (such as sugar, &c.) which, under ordinary conditions, are taken very much more slowly into the blood, and are soon decomposed when they get there. The extent to which the blood is contaminated and injured by this admixture, and also by the retention of urinary constituents, is not yet accurately known. "When we remember," says Dr. Parkes, "the great share taken by the blood-globules in the respiratory and heat-furnishing processes, it is scarcely possible to avoid concluding that their loss of salts is connected with the characteristic cyanosis and lower temperature in cholera. In *most* cases there is vomiting and purging before there is loss of heat, though this very soon follows in a slight degree, and then gradually augments. In other words, the diarrhoea coincides with the first or early chemical changes in the blood—the transudation of some of the constituents of the serum. The lowered temperature follows afterwards, at the time when we know that diffusion from the blood-cells into the serum must be taking place, and augments gradually as the diffusion increases." In all the cases examined by Dr. Marcus at Moscow, in 1832, the clot and serum evinced acid qualities on the application of litmus, except in four cases, where the discharges were watery and the reaction alkaline. The phenomena of the disease may thus be traced from the transudation of serum constituents as the starting-point. All the other chemical changes in the blood, and the most marked symptoms (such as the abnormal respiratory process), follow as a matter of course.

But the question may be also put in another form, as it has been already so well put by the able reviewer of Dr. Johnson's book in the *Saturday Review*,—"Why has the circulating blood stopped here (in the pulmonary arteries), and by what means has it been brought to a stand? Were the arrest of motion due to gradual thickening in consequence of the continued abstraction of its liquid portion, it would be found stagnating in the capillaries, as well as in the arteries. It must be borne in mind that one characteristic symptom of cholera—that symptom which, irrespectively of the





**Origin and Modes of Propagation of Cholera.**—One of the most interesting facts brought out by Dr. Farr and Mr. Radcliffe is, that

arteries, are doctrines which do not necessarily involve an acceptance of the 'elimination treatment' of cholera, nor presuppose any belief that cholera begins as a blood-disease. It is important that the different questions should not be jumbled together as one; particularly important now, because the notion of a primary blood-poison in cholera seems tending more and more to be superseded.

"1. First, as regards the state of the circulation in collapse. Personally knowing Dr. Parkes's great accuracy of statement, I attach (says Mr. Simon) the utmost importance to the descriptions contained in his work. And their details do certainly in great part justify the generalization which he makes of them.\* But whether the morbid phenomena which he describes are rightly accounted for by the doctrine of arterial obstruction in the lungs (either such as he supposes, or such as Dr. Johnson supposes) is matter of much more doubt: for feebleness of heart-contraction appears to be an invariable fact in choleraic asphyxia; and so far as this affects (or at least predominantly affects) the right side of the heart, so far it tends to produce much such a disturbance of circulation as would result from the supposed arterial obstruction. Present opinion seems, I think, generally to be that, in the main, it is the dynamical affection of the heart (not the supposed obstruction of pulmonary arteries) which gives the true explanation of Dr. Parkes's facts; but this would not of necessity imply, either for the pulmonary or for the aortic circulation, that all the arterial resistances are normal. Whatever etiological view be taken of the connection of the symptoms of collapse, it cannot be deemed unlikely that a much diminished volume and impaired fluency of the blood, when they have arisen, should excite certain phenomena of their own in the sphere of arterial contractility, as well as have their own physical consequences; nor, again, that certain changes of arterial tone should go with certain changes of cardiac action. Be this as it may, some of the phenomena presented in the aortic system in collapse are such as arterial contractility would seem very plausibly to explain. Such are some of the inequalities of temperature and circulation in the diseased body, not only as between internal and external parts, but as between different parts (external or internal) in the aortic circulation. Specially, for instance, I cannot conceive from what other basis to explain the tendency to equalization of temperature between external and internal parts which is apt to show itself in the fatal ending of collapse, and even to continue after death, as though a final relaxation of arterial rigidity permitted the blood at last to find way through its normal channels. And if the cold and cyanosed state of external parts in cholera be not to some extent under control of arterial contractility, I cannot conceive through what mechanism to explain that exceptional state of mammary circulation which permits the continued secretion of milk by nursing women who are in collapse.

"2. The belief that a primary 'blood-poison' is the proximate cause of cholera, the direct source equally of its intestinal and of its asphyxial manifestations, is, so far as I know, mere hypothesis. It has been much accepted as the only possible explanation of certain supposed, but very questionable facts in the natural history of the disease; specially in explanation of the supposed fact, that the utmost collapse of cholera may concur with little or no affection of the intestinal canal. It is of supreme pathological importance to be right in the matter of these premises. Is it, or is it not, true that choleraic asphyxia can arise otherwise than in consequence of the bowel disease? This question has been much perplexed, partly through the vast number of vague assertions which are current on the subject, and partly through an assumption which has often been prematurely, and perhaps wrongly, made, that the significance of the bowel disease in cholera is to be measured by the quantity of the fluid secretion from

---

\* "Yet it deserves notice," says Mr. Simon, "that even among Dr. Parkes's own cases of death in collapse the post-mortem evidence of interrupted pulmonary circulation was not universal; and I may add, though without attaching equal importance to the fact, that a citation of miscellaneous authorities on the state of the heart and lungs in death by collapse (such a citation as was given in 1833 by Prof. Phœbus in chapters v and vi of his classical *Leichenbefund bei der Orientalischen Cholera*) would show still less uniformity of evidence in that respect. Also, in my opinion, the assertion made by Prof. Griesinger (in § 483 of the admirable essay on cholera which forms part of his *Infections-Krankheiten in the Handbuch der Pathologie und Therapie*) deserves much weight, viz., 'that the distension of the right cavities of the heart appears not to be present during life, as percussion gives (invariably?) a small area of cardiac dulness.' Supposing the general accuracy of Dr. Parkes's descriptions to be conceded, judgment must, I think, be reserved on the meaning of the alleged exceptions. For, whatever question there may be as to the inter-dependence of the symptoms of cholera, it is certain that the disease, in proportion to its flux, tends to reduce more or less rapidly both the volume and the fluency of the blood; and till we know exactly what would be the ultimate anatomical expression of those changed physical states of the blood, taken by themselves, it is impossible to affix a right value to the cases of cholera where post mortem appearances, or facts observed during life, have not answered to Dr. Parkes's general description of the anatomy of death in collapse."



era, and diseases of a choleraic character, have been unusually fatal in the metropolis and England generally; that the mortality from

mysterious death too swift for remedy, which severe epidemics of cholera are singularly apt to produce. But, taking them at what they are worth, what reason is there to believe that the sufferers who were so stricken down had not bowel seizure as the ground of their collapse? No doubt the opinion has been current that cholera, acting in some mysterious way on the total organism, may 'kill and leave no sign;' but in proportion as exact morbid anatomy has been cultivated, that opinion has, I think, more and more seemed to rest on a mythical basis; and the doctrine of primary collapse ought at least, without hesitation, to be rejected for cases where post-mortem examination of the bowels has not been made. In illustration of these remarks, I would refer very particularly to the important case given by Dr. Sutton, in his report *On the Clinical Characters of Cholera*, 1866. It was a typical case of *cholera sicca*. It was a case of cholera death so swift that probably none of the reported 'upas poisonings' were swifter. But fortunately the body was anatomized. The whole length of the small intestine was found containing choleraic effusion; and to assume, in the face of that fact, that the cholera collapse was primary, would, in the present state of knowledge, be, to say the least of it, a simple *petitio principii*.

"In the present state of knowledge, then, I do not find it proven, nor do I see any theoretical convenience in taking for granted, that cholera begins as an active blood-change capable of producing primary collapse. The facts, so far as I know them, can all be reconciled with the belief that cholera begins as bowel disease, producible by direct contagion, without even a passive intervention of the blood, and that all asphyctic phenomena of the disease are supervenient sympathetic phenomena. That, so far as they are facts of cardiac paralysis and arterial contraction, they may be attributed to nervous sympathy between bowels and circulatory system, without reference to the greater or less humoral effects of the coincident flux from the bowels, is at present a tenable view. At the same time, I hesitate to accept as proven that cholera collapse is independent of humoral sympathy. That it may often be apparently so is, no doubt, well shown by the statements I have quoted from Dr. Goldbaum and others. But it must be remembered that in those comparative statements two most important variables are not taken into account. First, there is the varied rapidity of the local morbid process—a very considerable range of difference; and it is imaginable that the power of the intestinal flux to produce collapse may vary with the rapidity, rather than with the mere degree, in which it tends to inspissate the blood. Secondly, there is the varying susceptibility of the individual patient; and that this has range enough to account for very considerable differences of manifestation in the functions concerned in collapse will be evident to any one who has attentively studied the very kindred subject of febrile rigors. Indeed, the power of both the variables in question may be illustrated from that analogy; for all observers know how essentially the rapidity of the thermal rise is the determining condition for the rigor; and all equally know how one patient suffers rigor to the very verge of death from influences which would not appreciably disturb another one.

"In questioning the fact of a primary blood poisoning in cholera, I, of course, do not intend to deny that the blood *during* cholera is poisoned. From our earliest knowledge of the disease it has been on record that, when pregnant women have cholera, the intra-uterine offspring always invariably dies; and more recently, in proportion as the anatomy of the disease has got to be better studied, cases have accumulated, giving detailed evidence in support of an opinion which had from the first been entertained, that the infant in such cases dies of true choleraic infection. Waiving particular reference to earlier cases of this sort (for which see, for instance, Phœbus, 1833, op. cit. § 51,\* and Buhl, 1856, in the famous Bavarian report) I may quote some statements made by Dr. Goldbaum in the report to which I have already referred. In the three last epidemics, he says he has carefully anatomized twenty-two such infants, and never failed to find appearances which, collectively, he deems characteristic of cholera. 'In the stomach and upper part of the small intestines always there was a fluid like rice-water, sometimes a thick mass, consisting of exfoliated bowel epithelium; the heart was always ecchymosed; at the back of the tongue there were swollen papillæ, as there are in greater degree in adult cholera corpses; and in the kidney the yellowish cortex contrasted strongly with the blood-holding medullary substance.' It may, I think, be assumed for certain that the death of the

\* "Among the cases given in Phœbus's work is one where the infant was not actually born dead, but died an hour afterwards with all symptoms of the epidemic disease."



lubrious ; and, *second*, a susceptibility to the disease in the inhabitants of such places, produced by the habitual respiration of an impure atmosphere.

2. That the cause of cholera is a morbid matter which undergoes increase only within the human body, and is propagated by means of emanations (or discharges) from the bodies of the sick ; in other words, simply by contagion in the most limited sense of the term.

3. It is believed that the poison of cholera is swallowed, and acts directly on the mucous membrane of the intestines, and is at the same time reproduced in the alimentary canal, and passes out much increased, with the discharges ; and that these discharges afterwards, in various ways, but chiefly by becoming mixed with the drinking-waters in rivers and wells, reach the alimentary canals of other persons, and produce the like disease in them.

4. Assuming that the cause of cholera is a morbid matter or poison, it is probably reproduced in the air, as well as within the bodies of those whom it affects, and that its effusion may be due to the agency of the atmosphere.

5. It is believed that the cholera poison is increased by a species of fermentation, or other mode of reproduction, in impure, damp and stagnant air ; and it is maintained that it nevertheless is distributed and diffused by means of human intercourse, being carried in ships and other vehicles, and even in the clothes, especially in the foul clothes of vagrants, and the accumulated baggage of armies.

6. It is assumed that the material causes of the disease may be increased and propagated in and by impure air, as well as in and by the human body.

“Germany and England,” as Mr. Simon justly observes, “may claim between them the credit of having built up all the definite knowledge we yet possess regarding the pathology of cholera.” Griesinger and Hirsch of Berlin, Wunderlich of Leipzig, Pettenkofer of Munich, Thomé of Cologne, and Klob of Vienna ; combined with Orton, Tytler, Parkes, Greenhow, Snow, Sutton, Martin, Macpherson, Buchanan of London, and Dr. Andrew Buchanan of Glasgow, Budd, Baly, Gull, Marshall, Radcliffe, Lauder Lindsay, Thudichum, Sanderson, Beale, Farr, and Simon, are those who have most of all contributed extensive and definite observation under varied circumstances and numerous epidemics in this country and in India.

The microscopy of the body in cholera, and especially of the stomach and intestines, has led in Germany to the rediscovery (by Drs. Thomé and Klob) of microscopical bodies like *fungi*, innumerable and vehemently multiplying, whereof swarms are shed with prolific and infective power in each characteristic evacuation of the sick. At the International Medical Conference on cholera, which met at Easter of 1867, at Weimar, and which Mr. Simon attended, Professors Hallier of Jena and De Bary of Halle (two of the leading mycologists of Germany) were associated with Dr. Thomé and Klob to make, in common with them, a statement and appreciation of the facts which had been observed, and which are in substance that—“Both observers find in cholera evacuations and in the intestinal mucus of the dead body definite organic structures, consisting of excessively fine granules, clustered more or less densely in the inter-



of vegetable or fungoid origin. They appeared then to Dr. Parkes to be "but modifications of the same substance, namely, fibrine." He, however, recognized "vibriones in great numbers, and two or three oval transparent bodies placed end to end. When the stool was kept, these fungi increased in numbers" (Ob. 6). In the descriptions and plates of Klob, Thomé, and Hallier, the corpuscles and granular bodies which they figure are now recognized by Dr. Parkes as similar to those which so attracted his attention in 1849. He again noticed these bodies in 1865 and 1866, when they vividly recalled the previous observations to his mind. Dr. Parkes concludes from these and his own observations that this fungus development really exists, and invariably in cholera dejections. He found them in every stool which he examined in 1849 and in 1854; and he again saw them in 1865 and 1866. He is familiar with all the forms described by Klob and Thomé; and believes them universal in cholera stools; and they form the major part of the white flocculi of the true "rice-water stools" (*Report on Progress of Hygiene in Army Med. Dep. Reports for 1865*). In 1854, Dr. Lauder Lindsay also recognized large bodies which he named "gonidic," from their resemblance to the gonidia of the lichens. They appear quite globular, usually larger than pus-corpuscles; have a distinct wall, colorless and transparent; frequently a distinct central nucleus, also colorless, round which are aggregated a number of rounded granules of a bright greenish-yellow or orange color, resembling the chlorophylle grains in the cells of plants. These bodies have occurred in greater or less abundance in the evacuations of *all* the cholera patients under Dr. Lindsay's charge; and they pass through the digestive apparatus both of man and the dog apparently without change (*Edin. Med. and Surg. Jour.*, 1854).

To say that these fungi are the cause of cholera would as yet be premature; nevertheless their existence is capable of accounting for many of the phenomena of cholera, and especially its spread. The poisonous properties of fungi are well known, so that, apart altogether from the physical influence of such spores, by their development in the intestines, the products of their growth may be eminently poisonous and deadly. In this direction the cultivation experiments may yet be carried.

Professor E. Hallier, of Jena, whose reputation as a fungologist is not surpassed, has made numerous experiments by the cultivation of these spores; and from an able summary of his paper by Dr. Buchanan (*Ninth Report to Privy Council*, p. 512), it appears that his observations were made on the stools of a person ill with cholera at Berlin in 1866, and on the stools and vomita of a cholera patient at Eberfeld in 1867. The characteristic vegetable elements consisted of a fine fungiform matter which floated, and of more highly developed spore-cysts which sank to the bottom (Figs. 1 to 4. of engraved Plate). These spore-cysts were yellow or brownish bodies, consisting of a pale membrane inclosing highly refracting colored spores. The cyst-wall undergoes a series of changes, ending in its rupture or solution, when the spores become free. The spores then, by progressive (*uniparous*) partition (a process which may begin before



they leave the cyst), resolve into very small cells, grouped into balls and heaps, which Hallier calls "colonies of micrococcus." The small cells constitute the fungiform matter seen in the evacuations, and they attach themselves to any bodies there may be in the stool—to remnants of animal or vegetable food, to epithelium cells, or to oil-globules, when these and all nitrogenous matters to which these fungi cells fix themselves become of a dirty aspect and lose their structure.

Besides these two elements (the spores and minute cells), torula-like bodies (Fig. 3, *c*) were found in smaller number, and were shown by cultivation to develop from the micrococcus cells. The formation of these cells (which occur singly or in rows) marks a step towards a higher development of the micrococcus—towards the production of *oidium*\* forms of fungi.

In the stool and vomit from Eberfeld, the cysts and colonies of fungi were found in smaller proportion, and the free micrococcus cells more abundantly, and epithelium was seen in which the process of invasion by the micrococcus could be watched. The little cells fastened themselves upon the epithelium, and increased in size as the epithelial elements wasted. The parasitic cells of fungi always grow at the expense of any nitrogenous organic substance which they attack; and this is well seen in the fungus foot disease (*Mycetoma*) of India. (See end of Vol. I.)

To learn the development and ultimate form, so as to identify the fungus, Hallier had recourse to the artificial cultivation of the spores, on several substances on which *fungi* are known to increase, multiply, and grow, such as in sugar, starch, paste, flesh, and the like (Figs. 5 to 8 of engraved Plate), in the same way that our distinguished fungologist, the Rev. M. J. Berkeley, made out the nature of *Mycetoma*. In sugar, Hallier, conducting the growth at a temperature of 68°—88° Fahr., succeeded in growing from the spore-elements a long pale filament containing granular plasma, and divided by septa; from this elongated processes branched off, and the whole formed a structure greatly resembling the *oidium lactis*. This *oidium* plant bore at the ends of its branches bulbs (*macroconidia*, Fig. 7, *m*), either single or in shorter or longer series: if single, generally larger, and apt to develop *muror* forms; but if in series, more apt to develop *penicillium* forms. On the ninth day of the experiment some of the branches bore a cyst containing spores, pale and weakly. Only once did a well-developed colored spore-containing cysts make its appearance; and Hallier's experience led him to connect the absence of such cysts with the absence of nitrogenous matter from the soil in which the fungus was growing.

---

\* The *oidium* fungi belong to the order *mucedines*—the "blue moulds"—having flocci very short, producing a monoliform string of spores by tomiparous division. In temperate climates they grow on damp paper, dead wood, decayed fruit (grapes, oranges), on *porriqo lupinosa*, on honeycomb, on nettles, ground ivy, and on plants infested with *ergot*. They belong to the family *Hyphomycetes*, or "thready fungi," the characteristics of which are filamentous, with fertile naked threads, for the most part free, and bearing the spores at their apices. (See Berkeley's *Outlines of British Fungology*, pages 337 and 350.)



7

A variation of the experiment, by providing the fungus with starch paste, produced scarcely different forms; but a more interesting result came of the addition of a small quantity of tartrate of ammonia to the paste. For the first few days of this experiment nothing but *micrococcus* cells, and chains of similar elements resembling *leptothrix*,\* were seen; but about the fifth day there appeared a small brown speck in the paste at some little distance from the surface, at a spot where the reaction was alkaline. This brown spot gave to the microscope colored forms, filaments bearing *macroconidia*, single and in series (Fig. 7), and some of them also bearing bunches of spores, or well-developed cysts containing spores, and greatly resembling the cysts found in the original stools. Upon the occurrence of an acid reaction in the paste the growth of these bodies ceased.

The spores of the fungi were further grown upon muscular tissue immersed in sugar solution. The *micrococcus* cells were seen enlarging and budding, and developed the usual *oidium plant* with *conidia* at the ends of its branches. Shortly after numerous cysts appeared, which went through the same changes as the cysts in the original stool. The muscular fibres were invaded and decomposed by the *micrococcus*, just as the intestinal epithelium had been in the stool.†

A subsequent observation made with cholera fungi grown in Hallier's isolation apparatus, upon paste which had been boiled with tartrate of ammonia, gave highly developed cyst-formations

\* These belong to the family *Coniomycetes*, or "dust-like fungi," of which the spores are the prominent feature, and not the threads (as in *Hyphomycetes*), the spores being either solitary or concatenated, produced on the tips of generally short threads, which are either naked or contained in a perithecium, or compacted into a gelatinous mass. *Rust* and *mildew* are examples of this family. *Leptothrix* consists of the mycelial filaments of mildew fungi, abounding in foul water in flocculent masses.

† EXPLANATION OF THE ENGRAVED PLATE IN ILLUSTRATION OF PROFESSOR HALLIER'S EXPERIMENTS ON THE FUNGUS FOUND IN THE "RICE-WATER" STOOLS OF CHOLERA (p. 589 to 592 of text).

Figs. 1 to 4.—Vegetable Growths, of the Nature of Fungi, seen in the Cholera Rice-water Stools: Berlin, 1866.

1. Groups of swollen gelatinous spores in the act of forming *micrococcus* by repeated divisions of the nucleus. The spores are surrounded by *micrococcus* cells.

2. Gelatinous cysts swollen and breaking up. (c.) Small cyst with clear spores; (l.) gelatinous cyst; (h.) wall of cyst subsequent to discharge of the spores.

3. Yeast formations. (m.) A large colony of *micrococcus* corresponding with a spore; (b.) a large colony of *micrococcus* about to break up; (k.) a group of colonies originating from the spores of a cyst; (c.) *torula*-like cells grown from swollen and enlarged *micrococcus*.

4. (n.) Several cysts connected; (o.) a large globular cyst discharging its spores; (a.) semi-divided spore, showing the commencing formation of *micrococcus*; (b.) spore quartered; (c.) groups of spores from a small cyst.

Figs. 5 to 8.—Products of "Cultivation," No 1: Berlin Rice-water Stool of 1866, with Sugar Solution.

5. *Micrococcus* and *torula* formations.

6. Germination of *torula* cells.

7. Filamentous termination of a healthy germ. (m) *Macroconidiæ*; (c.) cysts; (d.c.) degenerated cysts.

8. Completely formed cyst with spores, one of which is just about to germinate, (k.)

Fig. 9.—(a.) Commencement of proliferation.



the development of the fungus outside the body. Thus in summer, and in summer only, in European latitudes could the fungus find in earth and night-soil the necessary temperature for its increase. The conditions for the production of *Urocystis* appear by the experiments to be, not high temperature only, but also a copious supply of nitrogenous with some hydro-carbonous nutriment, and a high degree of moisture. But besides these conditions the reaction of the fluid was found to be important, and this again to be dependent on the nitrogenous elements of it.

Hallier points out that the home of all penicillium-bearing fungi is Asia. The tilletia-bearing form occurs only on wheat, which is a plant imported from Asia. Hence he infers a further probability that the cholera fungus, which appears to be another development of the same species, is also originally Asiatic.

Further observations as to the precise effects of temperature confirmed the foregoing deductions. Fungus, while developing *Penicillium* only at ordinary temperatures, was grown upon appropriate nitrogenous soils at a temperature of 88° to 110° Fahr., and (when other circumstances were favorable) a development of cyst-forms took place from the budding of the *Penicillium*, precisely like the forms met with in the stool. A piece of intestine exposed to the action of the cholera fungus at this temperature got its epithelial elements rapidly destroyed by micrococcus. Converse experiments with low temperatures showed that the characteristic cyst-forms of cholera stools were not produced upon materials that were kept below 54° Fahr. The inference is therefore confidently drawn, that if the fungus be indeed the contagious material of cholera, cholera cannot maintain itself permanently in our latitudes. Other conditions under which the fungus did not grow were,—(1.) A temperature over 144° Fahr.; (2.) Sulphate of iron in concentrated solution; (3.) Carbolic acid (not the most potent agent of its kind in these experiments); (4.) Permanganate of potash; (5.) Wine (from its acidity, probably) and strong alcohol. Quinine had some influence, opium none, in preventing the destruction of animal tissue by the micrococcus. Of all experiments made to determine the power of chemical agents upon the fungus, chief success was obtained by the free acidification of the fluid. This is confirmatory of Pettenkofer's views upon the disinfection by acids of substances infected by cholera poison. Whenever the fungus grew in acid solutions it showed no cysts and no micrococcus, only penicillium and cognate forms. For fungus destruction on the large scale Hallier would give preference to sulphate of iron. But he insists particularly on the destruction of each individual stool before mixing it with other night-soil, and of course urges the systematic removal of all such matters to the field.

Professor Hallier's inquiry is next concerned with the circumstances under which the cholera fungus, indigenous to Asia, and only travelling into northern latitudes in the bowels of cholera patients, grows in its native soil of India. He recalls the fact that other forms of the fungus under consideration are peculiar to cereal plants, and that the *Urocystis*, with its characteristic cysts, inhabits



direct and circumstantial, each is also equally opposed by a "considerable number of obstinate facts." All of them, however, agree in two main points—namely, that cholera is induced by a special poison, and that this poison is of foreign extraction.

But, on the other hand, there are good grounds for believing that cholera has not in the present century for the first time appeared in this country, and extended itself over the greater portion of the habitable globe. The "*cholera morbus*" of Sydenham, prevalent in his time, and the "*griping in the guts*," or "*plague in the guts*," as recorded in the mortality bills of 1665, and described by Willis, and subsequently by Dr. W. Heberden, Jr., do not seem to differ in their essential phenomena from the disease supposed to be imported into this country from the East; although some believe that these descriptions of disease refer to dysentery, and not to cholera.

There are also abundant facts which seem to show that, under a different name, cholera was one of the most fatal epidemics by which the population of London was formerly afflicted. And there is no doubt that cholera, like every other epidemic disease, varies in its type, as it does in severity; for, if it is conceded that the diarrhoea so prevalent during an epidemic of cholera arises from the same cause, and is, in fact, the same disease in a different degree of intensity, as Orton showed in 1832, "there is as much variety in the aspect and symptoms of cholera as of scarlet fever; between the malignant cases of which and the extremely mild ones there is so vast a difference."

The principal differences shown by Mr. Radcliffe between the recent epidemic (1866) of cholera in Europe and those of former years are, (1.) For the first time in the history of the disease, Europe was invaded from the south. In 1829–32 and 1845–48 the disease spread from Persia to Russia, and thence along the Danube into Central Europe; and in Britain the towns first attacked on all previous occasions were sea-ports on the east coast. (2.) A remarkable feature in the epidemic of 1866 was its rapid and great extension along the coast line as compared with its slight and sluggish penetration inland. The central districts of Europe escaped altogether during 1865, except that there was an isolated outbreak in Saxony. (3.) The progress of the disease was much more rapid than in former epidemics. In 1829 cholera took fifteen months from the time of its entrance into Europe to reach Great Britain; two years, less one month, to arrive on the North American coast. In 1848 its diffusion occupied nearly the same period of time. In 1866 the disease had in less than five months spread from Alexandria to the coasts of the Euxine, and even to the western hemisphere. A strong point in favor of the view that in 1866 cholera was introduced from the Mediterranean by ships coming thence is furnished by the fact that Southampton was the only port at which ships arrived having had cholera deaths on board shortly before reaching England. Moreover, it had been predicted that the disease would enter the country by Southampton, and not, as before, by towns on the east coast; and the fulfilment of the prophecy will seem to many, as Dr. Parkes





medical relief (not restricted to paupers) were exercisable by local authorities throughout the country.

"On 18th July, from Poplar, the first cholera death in the metropolis was reported. Two days afterwards there was already an alarming proportion of cholera cases in parts of East London; and on the 21st the secretary of the London Hospital reported that the resources of that most useful institution were being overtaken by such claims for admission as attested a very terrible epidemic of cholera."

At the same time diarrhœa of so severe a form as to be called "choleraic" preceded the epidemic outbreak in 1865 and 1866; and if it is found impossible to disentangle at the beginning of an outbreak, cases of a *quasi* epidemic character from those of a *true* epidemic character, and to shut out absolutely a theory of the development of epidemic cholera by gradation out of quasi epidemic or severe diarrhœa, it is equally impossible to set aside the fact of exposure of the metropolis (and busy ports of embarkation and debarkation, like Southampton and Hull and Liverpool) to continuous transmission of the epidemic malady from the early autumn of 1865 to the early summer of 1866 (RADCLIFFE). The apparently distinct outbreaks in several towns and localities, however isolated, must all be regarded as forming parts of one general epidemic; and the histories of them, so ably set forth in the various reports collected together by Mr. Simon, Dr. Parkes, and others, compel the conclusion that the chief agents in the dissemination of the epidemic of 1866 have been the sick from the malady in its slighter, as well as more marked and characteristic forms—a conclusion which has been adopted absolutely of epidemic cholera by the International Sanitary Conference which met at Constantinople to consider the question of the preservation of Europe from this pestilence. The history of the epidemic in its entirety in this country points to the transmission of the disease to the metropolis and other ports from localities previously visited by it in Western Europe. Mr. Simon's dictum that "*contagious currents on the Continent of Europe must be deemed virtually current in England*," is to be accepted as an axiom in State Medicine, notwithstanding that links of transmission may fail to be discovered (*Ninth Report*, p. 288). The testimony of Dr. Macpherson also is to the effect that, whatever cholera we have had in Europe in former times, since 1817 at least it has been always carried out of India, or Persia, or Arabia, to other places.

"The outbreak of cholera in the metropolis in 1866 cannot well be considered apart from the wide diffusion of the disease on the Continent of Europe during 1865 and 1866. It is inextricably linked, both chronologically and etiologically, with that rapid dissemination of the malady which in May of the former year, commencing at the most sacred city of Mohammedanism, Mecca, extended to Egypt, and thence, before the close of the summer, to many places on the eastern and southern coasts of Europe, and in the basin of the Mediterranean. During the autumn the epidemic spread largely in the south of France and in Spain, appeared at Altenburg in Saxony (where it was introduced from Odessa),\* and ex-

---

\* *Die indische Cholera in Sachsen*, 1865; Dr. Rudolf Günther, p. 9.



houses, in which no change of air “seemed to take place for almost a week together.” According to the delicate and accurate observations of Mr. Glaisher, the meteorological phenomena of the three visitations of 1832, 1848, and 1854, appear to have been remarkably similar (excepting as to temperature). Indian medical officers, and those of the Black Sea fleet, give similar accounts as to the meteorological phenomena which attended the outbreaks of cholera, in their experience.

The chief meteorological phenomena of the epidemic period of 1865–66 have been summed up by Mr. Glaisher,\* and a comparison instituted between them and those occurring during the previous cholera outbreaks. The contrast is remarkable. The visitations of 1832, 1848, and 1854 were coincident with great atmospheric pressure, high temperature (except in 1832†), small diurnal range, (owing mostly to high night temperature), deficiency of rain, very little wind (and comparative stagnation of atmosphere and prevalent mist), a deficiency of electricity (indicated by the few electrical disturbances), and in 1854 “the presence of a remarkable blue mist,” which prevailed night and day, and total absence of ozone.

During the three months of principal prevalence of the outbreak of 1866 in the metropolis (July, August, and September), the atmospheric pressure was remarkably low. From the 26th of July to the end of the quarter the barometer, reading at the height of 160 feet, never reached the point of 30 inches—“a most rare occurrence,” as Mr. Glaisher writes.

The temperature of the air was low night and day, except in September, when the nights were warm. The daily range of temperature was small, “chiefly owing to low day temperature, particularly in August, and to a somewhat less degree in September, but the range in September was still further lessened by the high temperature of its nights.” There was an abundance of rain, and the air was in almost constant motion, “frequently blowing a much heavier gale than usual at this season of the year.” “Nearly all the circumstances,” Mr. Glaisher observes, “are directly opposite to those mentioned above as being present at the previous visitations of cholera, and have probably aided in checking its wider extension.” He adds, “One of the most remarkable atmospheric phenomena during the past quarter has been the prevalence of a peculiar blue mist, first seen by myself on 30th July, but which had been remarked by other observers in the preceding week. This blue mist since that time has been generally present. On some days no trace of the mist has been visible, and on other days it has been seen for parts of a day only. It has extended from Aberdeen to the Isle of Wight, and of the same tint of blue everywhere. This mist increased in intensity when viewed through a telescope; usually no mist can be seen when thus viewed; it increased in density during the fall of rain; usually mist rises after the fall of rain. Its density did not decrease when the wind was blowing moderately strong, but did decrease when a gale was blowing, but increased again on its subsidence. I do not know the nature of this blue

---

\* *Quarterly Return of the Registrar-General*, July—September, 1866, p. 18.

† *Appendix to Report of Committee for Scientific Inquiries—Cholera Epidemic*, 1854, p. 114.



sons. It is very plainly influenced most by the combination of influences known as "season" (MACPHERSON).

Such meteorological conditions have a marked tendency *to favor the chemical decomposition of organic substances, and to render the season defective in those atmospheric changes which, by decomposing and dispersing into space the products of decomposition, renew the purity of the air.* "The effect of temperature upon the Thames water is very remarkable in tainting the surrounding air, and is exhibited in the well-known fact that diarrhœa and summer cholera become prevalent among the inhabitants along the banks of the Thames after the temperature of the river has attained to 60°, and as the water declines from this temperature, so do these diseases in its vicinity." In Europe all the great epidemics have occurred in times of prolonged drought; and the dissemination or dispersion of the disease is very closely related to rainfall, as Dr. W. Budd, of Bristol, has shown. By diluting the poison, and by giving rise to floods which rapidly sweep it beyond the inhabited area, rain seems to have a powerful influence in checking the disease. But to have this effect the rainfall must be heavy and continuous—while, on the contrary, light and intermittent rains favor its spread.

The general result of all such observations is, "that whilst cholera may prevail within a considerable range of temperature, a moderately elevated one is most suitable for its development and propagation; and this, accompanied by a still, stagnant, and peculiarly oppressive condition of the atmosphere (more oppressive than the elevation of the thermometer can account for) and a moderate amount of moisture." With regard to the apparent anomaly as to temperature in the case of its outbreak in Moscow, and in the northern countries of Europe, such as in Sweden and Norway, it must be remembered that the internal atmosphere of the Russian, Swedish, and Norwegian houses is maintained at a high elevation during the winter months by means of stoves. It must be remembered, too, that the water used by the Russians in winter is often got from the melting of snow in the vicinity of the houses, and which snow is generally exposed to the reception of various excreta from the houses, just as the surface of the soil would be exposed. Hence the facilities for its spread in Russian hamlets.

Although meteorological statements appear to be a mass of confusion, from which we can scarcely deduce a single general principle; yet we know that organic germs, and seeds of various kinds, are capable of preservation under the most different and variable meteorological conditions; and also that particles or germs most microscopically minute are capable of actual demonstration in the air we breathe, as already stated at page 420. See also a most interesting and very suggestive paper on "*Germinal Matter and the Contact Theory*," by James Morris, M.D., Fellow of University College, London, 2d edition, November, 1867.

(2.) *Local causes.*—But, in order to give character and energy to the development of cholera, there are other conditions required besides those meteorological phenomena just noticed. These other conditions are described by Dr. Barton as the "*terrene element*," and



undergoes those developmental changes which is characteristic of its rapid dissemination as an epidemic. Such a condition of soil fosters the multiplication of the cholera poison. It does not generate the poison *de novo*, but by means of the evacuations of those sick of the disease the fermenting contents of choleraic intestines find their way into such soils; and the aptitude for the multiplication of the poison by specific fermentation is in accordance with a certain state of *humectation* of the soil, which again has a definite relation to the recession of the subsoil water after it has approached unusually near the surface.

But apart from the local conditions as to soil insisted on by Pettenkofer, there is very strong evidence to show that the decomposing choleraic discharges (already in a state of active fermenting change in the intestine of the cholera patient) will produce the disease—which will go on changing and multiplying a virus capable of spreading through the air as well as through the medium of water. In the outbreak at Southampton in 1866 there were instances both of transmission by water and by air (Parkes, in Mr. Simon's *Ninth Report on Public Health*, p. 253). The discharges need not pass into the ground to decompose or ferment there. They can decompose equally well in sewers, and, of course, can propagate their specific fermentation to the contents of such sewers, and such contents may find their way as gases or more material elements in the air or in the water.

*Impure water, lowness of sites, and the emanations arising from the decomposition of animal refuse, or of excreta*, are the local causes now satisfactorily determined to have a more or less constant connection with the development and propagation of cholera.

That *impure water* has a powerful influence over the intensity of cholera outbreaks is now unquestionably established by the observations of Drs. Acland, Sutherland, William Budd, Parkes, and the late Dr. Snow, and the specific inquiries of the Registrar-General and Mr. Simon. Yet still it is found that *impure water* is not a necessary element in the generation of the cholera poison, as shown in the report of Dr. Baly (pages 201–205), Budd as to Bristol, Pettenkofer as to Munich, and Günther as to Saxony. From their evidence, “cholera can do its very worst where the drinking-water can play no possible part in its dissemination” (*Brit. Med. Jour.*, April 13, 1867, p. 416).

The localized attacks at Theydon Bois in 1865, in the east of London and in Southampton in 1866, all point unequivocally to impure water. With the *general outbreak* at Southampton, however, impure water had nothing to do. It had only to do with the production of the disease on board the steamship “Poonah” just before her arrival from Gibraltar, where she took in a tankful of very foul water, which, from its peculiar smell, evidently contained sewage (PARKES).

As regards London, it has been shown by Dr. Farr that *the elevation of the soil* has a more constant relation to the mortality from cholera than any other known element, the mortality from cholera being in the inverse ratio of the elevation. Yet, like the condition





breathed with the '*privy odor*,' and that immunity from this appeared to secure immunity from cholera." The observations of Mr. Orton and of Dr. Greenhow are confirmed by the investigations of Dr. Pettenkofer at Munich and at the village of Gaimersheim. Dr. Barton, of New Orleans, Dr. Milroy in his report on the epidemic at Kingston, Dr. Buckler in his account of the outbreak in the Baltimore almshouses, and Dr. Parkes at Southampton, give similar evidence confirmatory of the injurious influence of the *fermentive decomposition of animal excrement*.

The outbreaks of cholera in some of the camps in Bulgaria and the Crimea, especially at Aladyn and Alma during the war, also furnish sufficient illustrations; and I believe the outbreak of cholera at Scutari, in November, 1855, which suddenly commenced in the camp of the Osmanli Horse Artillery, had a similar origin.

*Propagation of Cholera by Human Intercourse.*—When cholera appeared in its epidemic form in this country in 1831, the majority of European practitioners were decided contagionists. Subsequently to that period a reaction of opinion occurred, and the question was discussed for many years without any definite result. In 1848, when the disease again became epidemic, many of the higher authorities coincided with "the solemn declaration of the Board of Health, that the malady was not in any way contagious, and that no danger was incurred by attendance on the sick." "A large body of evidence, however, now renders it certain that human intercourse has at least a share in the propagation of the disease, and under some circumstances it is the most important, if not the sole, means of effecting its diffusion" (Dr. BALY). Very positive evidence has now accumulated in abundance to prove the transmission of the disease by human intercourse. Healthy men carry the disease with them by their clothes, by their ships, and by their caravans. That such is the case, we have now ample evidence in the *Bengal Report*, of 1824, by Dr. Jameson; in cases related by Mr. Orton, in 1832, and by Dr. J. Y. Simpson, in 1838; in *The Edinburgh Monthly Journal* for 1849, by the late Dr. Cruickshank, at Dalmellington, in Ayrshire; by Dr. William Robertson, detailed in *The Edinburgh Monthly Journal* for August of that year; and more recently the account of the outbreak at Arbroath, in Scotland, in 1853, by Dr. T. Trail; and cases by Dr. Alison, in 1854, in the paper already noticed; in the report of Dr. Berg, of Stockholm, in 1848; in the Norwegian Reports of 1850–53; in the Report of the College of Physicians of London, in 1854, and the several reports of 1865 and 1866. These records afford undoubted instances which show that human intercourse is occasionally influential, in some way, in transmitting cholera into detached localities, where it may seize upon two or more individuals, and then cease. But it is no less certain that its general extension over the world cannot be accounted for by human intercourse alone, to the exclusion of aerial contamination. It is curious that in India, the birthplace and headquarters of the disease, the doctrine of contagion is almost universally disbelieved in by our professional brethren. The opinion generally entertained in India is opposed to the doctrine of contagion (Morehead, *Indian Annals of Med. Science*,



2. By means of bed and body linen, and other articles tainted with the rice-water discharges.

3. Through the medium of the soil. The discharges being liquid, the great bulk of them find their way to the ground, from which the poison may be propagated in three ways,—(a) By rising into the air as a product of evaporation; (b) By percolating into the drinking-water; (c) By atmospheric dispersion in the form of impalpable dust, after it has passed into the dried state. It is, of course, difficult to establish these modes of propagation by direct proof; but circumstantial evidence, and evidence by analogy, is so cogent and weighty, that no reasonable doubts can now be entertained regarding these modes of propagation. By *experiment* the *enthetically* contagious poisons (*e.g.*, vaccine variola, woorara, &c.) are known to retain their properties in a dormant state for indefinite periods of time after having been dried up, and to recover these properties again when moistened. Evidence almost as certain as experiment demonstrates the same regarding the poison of scarlet fever, malignant pustule, glanders, and syphilis. Therefore, it is probably true of cholera, and the more so that the numerous and well-authenticated instances of the propagation of the disease through articles of dress shows that the poison, during its transit, must necessarily have been in a dried condition—a condition which entirely protects organic bodies from certain molecular changes; so that, so long as the material-holding poison remained in this dry state, no definite limit could be stated as to how long the morbid agent might retain its specific powers. From this point of view a single case may give rise to a wide-spread infection; and as cases multiply, it becomes more and more impossible to trace their lineal succession.

The relative share which the modes of propagation (here indicated by Dr. Budd) take in the propagation of cholera must vary with season and climate, with temperature, with the habits of the people, with the nature of the soil, with the water-supply, with the prevailing wind, and with general sanitary arrangements.

The experience of 1866 and of 1865 confirms all previous experience as to the propagation of cholera, so well summed up by Mr. Simon in his official memorandum of July, 1866. In it he assured the public that cholera is so little contagious, in the sense in which small-pox and typhus fever are commonly called contagious, that, if proper precautions are taken where it is present, there is scarcely any risk that the disease will spread to persons who nurse and otherwise closely attend upon the sick. But he admits it is not less true, that all matters which the patient discharges from the stomach and bowels are infective; that the patient's power of infecting other persons are due entirely, or almost entirely, to these discharges; that these, however, are comparatively non-effective when first discharged, but afterwards, while undergoing decomposition, acquire their maximum of infective power; that, if cast away without previous disinfection, they impart their own infective quality to other excremental matters; that if they get access, even in the smallest quantity, to wells or other sources of drinking-water, they may



[Cholera prevailed extensively in the United States army during the year 1866. There were 2813 cases, and 1269 deaths. It appears from Dr. J. J. Woodward's report to the Surgeon-General (*Circular No. 5, War Department, S. G. O.*, 1867), that it spread over the country during that year, extending as far westward as Forts Leavenworth, Riley, and Gibson, and in the southwest to Texas. In its progress it followed the lines of travel rather than any general westward course, and, in the case of the army, it especially followed the movements of bodies of recruits. The epidemic, so far as the army is concerned, evidently radiated from two chief centres—New York city, and Newport Barracks, Kentucky. The first reported case in the army, in 1866, was at Fort Columbus, Governor's Island, New York Harbor, in a recruit from the rendezvous at Minneapolis, Minnesota, of whose previous history and exposure nothing was known; he had been but three days at the post. In about an hour after his admission into the hospital another case occurred, also a recruit, with previous history unknown. Cholera was at the time prevailing in New York city. The fort too was in the immediate neighborhood of a quarter of the town chiefly infected, through which recruits passed with more or less delay. There was besides frequent daily communication between the fort and the town. Recruits from Governor's Island carried cholera to Hart's Island, another recruiting depot on the East River. The infection spread by readily traceable steps to Georgia; to Louisiana, by way of New Orleans; to Texas, by way of Galveston; to Louisville, Kentucky; to Richmond, Virginia; and to La Virgin, Nicaragua Bay. From Richmond it was carried to Norfolk, Virginia; from Louisville to Bowling Green, Kentucky. The probabilities appear to be that the disease was carried from New Orleans up the Mississippi River to various points on that stream, and west of it; and though the whole chain of evidence is not complete, yet there is a sufficient number of known cases of the transfer of the epidemic from one post to another in this region, to put this view of the whole movement beyond reasonable doubt.

The other principal centre appears to have been Newport Barracks, Kentucky, where the disease was plainly introduced from the infected city of Cincinnati, on the opposite side of the Ohio River. Although it did not prevail to any great extent at this post, yet it is in evidence that it was carried thence to Augusta and Atlanta, Georgia, and to Nashville and Memphis, Tennessee (WOODWARD, *l. c.*)

The following instances of the portability of the disease are cited from the official report.

1. On the 14th of July the steamship San Salvador left New York with seventy or eighty cabin passengers, and sixty in the crew and steerage. She touched at Governor's Island and took on board 476 recruits for the Seventh United States infantry. The men were lodged between decks, and were greatly overcrowded. On the second day out cholera appeared among the recruits, and when the vessel arrived at quarantine, near Savannah, Georgia, three deaths had occurred, and there were twenty-five ill of the disease. The troops were landed on Tybee Island. Cholera continued to prevail on the island during July and the first few days of August. Altogether there were 202 cases, and 116 deaths. The cabin passengers and crew of the San Salvador appear to have escaped, but of the ten white citizens residing on Tybee Island, nine were seized with cholera shortly after the arrival of the infected ship and five died. The tenth fled from the island, and is reported to have died of cholera some-



Those who argue against the availability of quarantine as positively protective against the dissemination of cholera, assert that it has never succeeded, and never can. The difficulty lies in making the means absolute. In proportion to its strictness is the risk from infected sources lessened. It is relatively, if not positively protective, and this view is fast gaining ground, as the result of a large body of facts. If efficient, it stops one mode of diffusion, and that a pretty potent one.

But admitting all that is claimed and probable of the dissemination of malignant cholera by human intercourse—body emanations, gastro-intestinal discharges, infected clothing—it is equally certain that, to account satisfactorily for its spread at times, we are obliged to own the agency of the atmosphere as a carrier, or an epidemic constitution, as well as the influence of localized conditions determining outbreaks and intensity. During the first two months after the French army landed in the Crimea, it lost more men by deaths and invaliding from cholera, than from gunshot wounds, from the battle of the Alma to the fall of Sebastopol; yet it brought no cholera with it, nor was there any at the time of disembarkation. The outbreak seemed to be due solely to atmospheric causes and insanitary conditions.

The organic theory as a cause of epidemics, first broached by Kircher, sanctioned by Linnæus, and ably advocated in late years by Sir Henry Holland, Henle, Dr. J. C. Nott, and others, was applied to cholera in Great Britain in 1849 and 1854, but, unsupported by observation, made but little headway. Recently there seems to be a tendency to a reconsideration of this hypothesis, which is certainly a very attractive one, and which would, if demonstrated by physical evidence, offer an easy and satisfactory solution of many of the mooted points surrounding the dissemination of cholera. "Many of the phenomena observed during the march of cholera epidemics," writes Mr. Goodeve, "might be explained much more satisfactorily upon the supposition of the exciting cause being masses of organisms moving in obedience to atmospheric impulses and currents, than by most other theories. They might multiply wherever they found a fitting nidus, which might be in privy atmospheres, or in air abounding in emanations from decaying and putrefying matter, or in crowded rooms, and, indeed, in all vitiated atmospheres. They might appear to impart an infecting character to the choleraic discharges by multiplying enormously in them" (Reynold's *System of Medicine*, vol. i, p. 147). Dr. Henry Hartshorne (*Cholera, &c.*, 1866) is a decided advocate of the organic theory, and he believes that the cause of cholera is a (yet undiscovered) protozoon, or primal organism, of extreme individual minuteness, which, on entering the human body, affects it as an organic poison; and that the conditions which favor and maintain in life multiplication and migration, this *ens primalis*, are afforded by animal matter in a state of rapid and foul decomposition, along with moderately high temperature and ordinary moisture.

Dr. Lionel S. Beale, for some time engaged in the study of the poison of contagious diseases, has, as the result of minute microscopical inquiry, reached these conclusions: (1.) The contagious or infecting principle consists neither of insects, of animalcula, nor any kind of vegetable organism. (2.) But of living matter formed in the organism of man or

---

Clellan, Assistant Surgeon United States Army, from which it appears that cholera broke out at various points in the vicinity of Fort Delaware, in fact, encircling the post, but did not invade the garrison, although one case, which recovered, occurred in the family of an officer on the island." (*Circular No. 5, 1867.*)





times. There may be from ten to fifty copious watery stools, and frequent copious vomiting, before there is any great loss of heat and failure of circulation. When purging commences twenty-four hours, or two or three or four days, before the violent symptoms, such as vomiting, purging, or cramps, such patients are said to have "premonitory diarrhœa." But there is always some degree of loss of heat and failure of circulation even in the slightest cases, else the case would be mere watery diarrhœa, attended only by exhaustion, and not by the symptoms peculiar to cholera. Cramps are seldom present till the stools put on the true choleraic character—viz., of copious white flocculi suspended in a watery fluid. The algide symptoms come on gradually, and are less intense than in the following forms; recovery is also more common. In the recent epidemic in London (1866), Dr. Sutton gives forty-one examples in which there was undoubted premonitory diarrhœa, the duration of the diarrhœa being as follows: In three cases, 12 hours; in one case, 18 hours; in one case, 19 hours. In seven cases, 1 day; in one case, 1 day and 9 hours; in twelve cases, 2 days; in six cases, 3 days; in in two cases 4 days; in two cases, 5 days; in one case, 6 days; in one case, 7 days. In two cases, 2 weeks; in one case, 5 weeks; and in one case, 8 weeks. In more than half the number of cases the diarrhœa preceded the marked symptoms by one, two, or three days; and of the forty-one cases, the diarrhœa in twenty-six was limited to the first three days.

2. If the poison acts with greater intensity, we have the second variety, in which there is less physical alteration in the fibrine, and the circulation is carried on for a longer time. Consequently, the characteristic change is not evidenced solely or chiefly in the interior of the vessels; but is partly transferred to the exterior of the vascular system. The albuminoid constituents, fibrine, and perhaps albumen, are effused in large quantities, and in all parts of the body, though chiefly on the free surfaces of the skin, alimentary mucous membrane, and more rarely the bronchial mucous membrane. The general nature of this effusion forms two characteristic distinctions between cholera and diarrhœa; for diarrhœa is a disease confined, in the first instance, to the eliminating part—viz., the large or small intestines, as the case may be—and is unattended, as a general rule, by the effusion of albumen and fibrine. The worst forms of this variety are seen in those cases in which, after two or three choleraic stools, severe and long-continued cramps come on, accompanied and followed by intense algide symptoms: after death the small intestines are generally found distended with the thick, white, flaky substance. Other cases of this variety present infinite modifications in severity, according as watery elimination is added to effusion of the fibrine; in other words, according as they tend towards the slighter forms.

3. Thus, if the final change at once occur, and there is a complete and rapid arrest of the circulation, either from the intensity of the cause or from constitutional predisposition, the worst variety is produced, in which "a mortal coldness comes on from the beginning." As the circulation is soon almost entirely arrested by physical alter-



orbit; every feature, moreover, is sharp and pinched, as after a long wasting disease; the complexion thick and muddy; the lips and tongue purple. All these great changes have been known to take place in a few minutes.

In addition to this sad state, the vomiting is constant, the purging most incessant, and the pulse, though often natural, sometimes rapid, yet in some cases it is not to be felt, even from the first moment of the attack, either in the large superficial arteries or at the wrist; the voice is strangely altered; its firm and manly tone changes to a low, feeble, and unnatural sound. The urinary secretion is likewise entirely suppressed, while no bile flows into the intestines. The only organ which seems to preserve its powers is the brain; and the patient often to the last moment of his life retains the power of thinking, and of expressing his thoughts distinctly, sometimes full of hope, while at other times he seems indifferent to the fate which too often inevitably awaits him.

The symptoms characteristic of the collapse stage during the late epidemic (1866), according to Dr. Sutton, in the Cholera Hospital and in the London Hospital, corresponded with those witnessed in other epidemics. "The pulse was only just perceptible—that was with great care—or the patients were pulseless; the extremities were cold; the tongue was very cold, sodden, coated with thin white fur; marked lividity especially in adults; old people and infants were, as a rule, less livid; the voice was reduced almost to a whisper; the eyes were sunken, pupils dilated, conjunctiva white and glassy; hands sodden and shrivelled; the patient restless, turning from side to side, with the eyes for the most part wide open, or closed only for a few moments at a time; very wakeful; excessive thirst, to such a degree that little children would get out of their beds and go and place their mouths under the water tap; cramps in the calves of the legs, extending up to the thighs and walls of the abdomen, in exceptional cases into the upper extremities. The patients manifested a great indifference as to their condition. When the patients were in extreme collapse the purging often ceased, and that in some cases for some hours.

"In the worst cases of cholera the vomiting and purging began suddenly and violently, went on rapidly, the algide symptoms set in very early, and there was very little and often not any purging during collapse" (*Ninth Report on Public Health*, p. 381).

On the accession of the spasms, the vomiting, and the purging, the disorder is fully developed, and the crisis is at hand, which in a few hours must decide the fate of the patient. The termination may be favorable or unfavorable: if unfavorable, he may die with all the symptoms just narrated strongly marked; or, should it be favorable, they may abate, and a happier prognosis be formed. Unfortunately, however, it too often happens that, although the stomach retains what is taken, and the purging appears checked, and the patient falls into a doze, yet the weakness, the entire cessation of the pulse, the coldness and lividity of the surface, and the ghastly expression of the countenance, show that a few hours must close the scene, often with so little struggle that death is only marked



not less fatal than, typhoid fever. These *typhoid* symptoms, common in Europe and America, are said to be unknown, or nearly so, in India, where, if a secondary fever ensues, it assumes the form of the remittent fever of that country. But remittance is characteristic of typhoid fever; and this character may only be more expressed in India than in Europe. For the first few hours after the febrile reaction commences the tongue is white, but it quickly becomes brown and dry, while black sordes incrust the teeth and lips. The eye becomes deeply injected and red, the cheek pale or flushed, the pulse rapid, and the temperature of the body a little above the natural standard. The patient, either delirious or comatose, then lies in a state resembling the last stage of the severest typhoid fever of this country. This struggle usually lasts from four to eight days, when the symptoms either gradually yield or death ensues. In a few mild cases the fever assumes an intermittent type, or sometimes a quotidian, sometimes a tertian form: all these cases usually recover. Such is a general outline of the symptoms of this formidable disease.

The blood in cholera varies according to the stage of the disease. In the cold stage it is usually of an unnaturally dark color and thick consistency, so that it flows with difficulty from the veins, and very imperfectly separates into clot and serum. Blood taken from the temporal artery has been found equally black and thick. After the secondary fever is formed, the quantity of serum increases, till at length it is much more abundant in the blood than natural; and it is singular that this takes place notwithstanding that the secretion of urine is re-established.

**Duration of the Disease.**—It is of importance to determine whether the cholera process is limited in its duration. Dr. Sutton has attempted the observation; and finds that while the “cold stage” is always present more or less, the “hot stage” may be absent, and is no essential part of the disease. It varies very much when present. In mild cases it is very short and scarcely appreciable. In severe cases it is long and protracted, at least in this country. The phenomena of collapse appear not to be limited to any definite time. Some patients became collapsed very early; others not until vomiting and purging had continued several hours, and was then often *not* protracted. In the milder cases the algide symptoms were scarcely, or even not at all, marked; and all experience has shown that the collapse of cholera is not always present.

Dr. Sutton then endeavored to ascertain what period elapsed from the time when a patient was seized with the characteristic symptoms of the cold stage—the violent symptoms—to the time of his entering reaction. He finds there are good reasons for believing that the *cholera process* runs a definite course of from twenty to thirty hours.

**Relation of Vomiting and Purging to Algide Symptoms.**—When patients went into collapse the vomiting and purging very greatly diminished, and in some cases entirely ceased; and some of the worst cases—cases which seemed almost sure to prove fatal—had very little, and often not any, purging. Thus cases characterized by the most continued purging and vomiting were not by any means



color and a greasy perspiring skin and a coated tongue, he will in all probability pass bloody evacuations, and then will certainly die. Dr. Sutton thus recognizes a class of cases having the following symptoms: The patient is seen lying on his back, eyes open, looking very wakeful, mind collected, voice weaker than natural, at times the typical choleraic voice, color natural, lips natural, complexion greasy, tongue sometimes cold, livid, of gray color and covered with white fur; at other times the tongue is warm and coated with yellow fur. The hands are of a livid red color, cold, and shrivelled. The temperature in the axilla is generally lower than usual. Respiration is labored, and generally accelerated—often 25, sometimes 40, a minute. The pulse at the wrist may be only just perceptible, and very often such patients are pulseless; there may be no purging for hours together, and very little vomiting. There may be profuse perspiration, the face and hair wet with it. The patient may lie for hours like this, and even one or two days. Such bloody evacuations appear on an average about twenty-eight hours after the violent symptoms of cholera set in.

When the algide symptoms are most severe the reaction is greatest and most protracted. The longest reaction was seventeen days, the shortest sixty hours, in Dr. Sutton's experience. In the mild cases the longest reaction was seven days and the shortest twelve hours. The duration of suppression of urine is also in proportion to the severity of the algide symptoms. In one case no urine was passed, and none discovered in the bladder, for six days and ten hours; in two cases none for five days, and in two none for four days. In the milder class of cases three days was the longest period of suppression, and the shortest ten hours.

**Chemical Changes undergone by the Body in the Progress of Cholera.**—Dr. J. L. W. Thudichum, at the instance of Mr. Simon, made important observations during the epidemic of 1866, which are published in Mr. Simon's report of that year. The following is a summary of his results:

The blood after death during collapse contains urea in variable abundance. The rice-water like evacuations contain butyric acid, and yield nitrogen and carbonic acid, but no urea. In bodies dead at an early period there was no urea; and more seemed to accumulate after a protracted algide stage, and much more after three to six days' torpid condition; and the greatest amount of urea was found after a long algide stage, with rise of temperature at the end.

*The secretion of bile* is completely arrested; and in extreme cases a clear white fluid percolates through the hepatic ducts, free from bile, coloring matter, and albumen. It seems to be simply water, with a trace of alkali and a vestige of mucus. In some instances the fluid is colored, but contains no bile acids. The bile ducts shed their epithelium.

*The blood loses water, albumen, and salts*, and is incapable of passing the capillaries with the usual freedom. It retains most of its coloring matter in its normal chemical composition; and Dr. Thudichum's observation led to the conclusion that any fermentation of





were obtained by Dr. Lauder Lindsay in 1853. The evacuations are in an active state of decomposition, and evolve gas, which at first is composed almost entirely of nitrogen; soon, however, carbonic acid prevails, and ultimately nothing but carbonic acid is evolved. At one period some hydrogen is developed. In 1848 Dr. Parkes examined many cholera stools, and his observations coincided with those of O'Shaughnessy, Vogel, Wittstock, and Andrew Buchanan (of Glasgow). The thin fluid was always alkaline, and contained an abundance of alkaline chlorides, phosphates, and sulphates, and a certain proportion of albumen. The *odor* was always peculiar.

Dr. Thudichum cannot discover any specificity in the above ingredients; but many of them are analogous to the products of ordinary processes of putrefaction. If it is admitted that the cholera evacuations acquire infective powers only after a period of fermentation, it is also easy to understand that the specific infecting power may belong to albumen or mucine at a particular stage of disintegration or chemical cleavage. The next knowledge which it is necessary to acquire is evidently this—namely, the exact period at which the rice-water stools acquire infective properties, and their chemical composition at that period. The most dangerous period of the choleraic stools is believed to be when they become very ammoniacal. This occurs usually immediately they are passed, but not to any extent for some time; and anything which makes and keeps them acid prevents the ammoniacal change (PARKES).

*Urine in Cholera Reaction.*—As the complete suppression of the urinary secretion in collapse, lasting for hours or days, is one of the most striking and peculiar features of cholera, so its reappearance is amongst the earliest and most auspicious signs of beginning recovery. The first secretion mostly contains the evidence of the mechanical obstruction of the minute channels of the kidneys, and of the general death of the epithelia of the urinary passages. It also contains the sign of continued resistance to the blood-current through the kidneys in the form of transuded albumen of the blood. And in many cases it carries small quantities of peculiar abnormal ingredients, which may perhaps be products or remnants of processes engendered by the choleraic process in the blood. The quantity is at first very small—urea much diminished.

[*Temperature in Cholera.*—Though observations on this point were made in 1831, the first which merit attention are those of the epidemic in Europe of 1848–50. But the statements of observers differ widely, for whilst Von Bärensprung and others assert that there is in the algide state a general fall in the temperature of the body, that the loss of heat is not only external but general (Müller's *Archiv*, 1852), Zimmerman, from two observations, came to the opposite conclusion (*Deutsche Klinik*, 1856). Briquet and Mignot believed that the greatest fall of temperature took place in the algide state, though they admitted to having in a few cases found a rise. Similarly opposed statements were made by others. All these measurements, there is reason to suppose, were axillary, and, therefore, of little value; for in the algide state the temperature of the axilla is considerably lower than that of the natural outlets, and it is



perature of the whole body. (8.) During complete convalescence an abnormally elevated temperature is frequently ascertained, without any apparent pathological cause (Virchow's *Archiv für Patholog. Anat. und Physiol.*, Jan., 1867; *Glasgow Med. Jour.* May, 1867.)]

*The Temperature in Cholera*, as determined by Dr. Thudichum, falls steadily from normal to  $5.4^{\circ}$  Fahr. or  $7.2^{\circ}$  Fahr. below it, and in most cases very rapidly.

The lowest temperature is quickly reached in deepest collapse; and the minimum temperature of all cases observed in the algide stage are below the lower limits of the fluctuation of health. The maximum temperature of the majority of cases observed are below the upper limits of the fluctuation of health.

The lower the temperature and the longer the duration of the algide stage, the higher and the longer continued is, on the whole, the temperature of the tepid stage, which does not exceed the upper normal, unless the temperature of the algide stage had previously sunk below  $95^{\circ}$  Fahr. But the temperature may for a short time reach  $95^{\circ}$  Fahr. or less, and yet the temperature of the tepid stage not rise above the upper normal.

When the maximum temperature of a case of cholera remains throughout below the normal average, the case will probably be fatal. Among the thirty-nine cases observed, all such cases, seven in number, proved fatal.

On the basis of the thermometric observations alone, cholera may be divided into two stages,—the first or algide stage, from the beginning of symptoms to that period where temperature reaches again the normal limits or average; and the second or tepid stage, in which temperature either remains within the normal limits or rises more or less above them, in some cases even to febrile height, afterwards descending again to normal limits.

But on the basis of all the pathological phenomena and clinical data, the following seven stages of cholera may be distinguished (THUDICHUM): (1.) *Fecal diarrhœa*; (2.) *Choleraic diarrhœa and vomiting*, quick sinking of temperature; leading to (3.) *Asphyxia or collapse*, in which lowest temperature is reached; (4.) *Reaction*, which may be defined as the cessation of collapse and the beginning of the re-establishment of the suppressed functions; (5.) *Torpid stage*, or secondary period of algide stage, in which, reaction notwithstanding, temperature remains below the lower normal limits, and then gradually or suddenly rises to the normal average; (6.) *Tepid stage*, in which, during continued reaction, temperature rises to normal or its upper limits, more rarely somewhat above; (7.) *The febrile stage*, only reached in cases where the entire algide stage has been very long, or where there are complications, or secondary lesions arising out of the choleraic process. Reaction does not always terminate the algide stage. For although, from the moment of the beginning of reaction, temperature rises somewhat in most cases, in exquisite cases it does not reach the lower limits of normal fluctuation. The algide stage is evidently continued into the state of reaction, and the tepid stage is the result only of continued reaction.



ries." It is not yet determined whether the general temperature of the blood is not febrile.

As regards temperature, the following conclusions were arrived at by Surgeon A. Leith Adams and Assistant-Surgeon F. H. Welch during the epidemic at Malta, of which they have given so admirable a report (*Army Med. Dep. Report*, vol. vi, 1864, p. 341):

"1. That a strongly marked, rapid downfall from the average normal temperature,  $97^{\circ}$ , takes place soon after the setting in of the cholera symptoms, and the extent in proportion to the dose of the poison; the downfall being characterized in the healthy young, and up to middle age, by elevations and depressions, each succeeding one of the latter reaching a lower point than the preceding one; in the aged, weak, or debauched constitution by an uninterrupted sinking of the thermometer. The average fall from normal temperature into collapse was  $11^{\circ}$ , the extremes  $7^{\circ}$  and  $15^{\circ}$  Fahr.

"2. That the highest temperature at which the general symptoms of collapse became apparent was  $90^{\circ}$ , the lowest  $82^{\circ}$ , the average  $86^{\circ}$  Fahr.

"3. That during the period of collapse the temperature underwent but slight variations in the aged, weak, or debauched; while in the young, and up to middle age, it was characterized by undulations.

"4. That the stage of complete collapse is not marked by any characteristic unvarying point of temperature. It would seem that an excessive dose of the poison is accompanied by a corresponding loss of heat; but when the vital stamina is deteriorated by drunken habits or delicacy of constitution, either the general symptoms of collapse are present when the thermometer makes no great fall, or an excessive lowness is reached with no corresponding general indications—*e. g.*, Cases 1, 2, 6, and 13 [in their Report]; while, on the other hand, a hardy well-used constitution does not betray signs of failing until the respiratory function is much interfered with. The lowest point reached during life was  $73^{\circ}$ .

"5. That a general brightening up of the patient, unaccompanied by any change of temperature, often preceded the final downfall, and was exceedingly deceptive until appreciated rightly.

"6. That the general signs of reaction were preceded by a marked elevation of the temperature, and when convalescence ensued, this reaction was characterized by fluctuations tending towards reinstatement of normal temperature. The average rise from complete collapse into full reaction was  $6^{\circ}$ , the extremes  $8^{\circ}$  and  $4^{\circ}$ .

"7. That when death ensued, whether preceded by reaction or not, the fall of the thermometer was most marked and rapid.

"8. That after death a rise of temperature ensued in the cases of great severity and quickness of course; but when the disease was prolonged, the patient falling into that senseless condition well expressed as 'death in life,' the contrary was the rule.

"9. The readings of the hands and epigastrium followed the breath's variations, though not always in the same ratio. As will be seen, the epigastrium especially showed a great tardiness in assimilating itself to the others, and was very tenacious of its heat.

"Thus, the thermometer indicated that in the aged and delicate the vital powers gave in to the poison, step by step, commensurate with the dose; collapse reached, a comparative quietness ensued, followed by the system asserting its superiority, or succumbing rapidly. The course from the onset to the termination was gradual, with no marked deviations.



army, also, it appears to have been universally observed that the officer suffered in a less proportion than the soldier, the cavalry than the infantry, and the infantry less than the hard-laboring ill-fed camp-follower.\* The troops on march likewise universally suffered more than the troops in quarters; and this influence of long marches appears to indicate something more powerful than mere fatigue in bringing about the disease. Dr. Balfour has proved that of the native soldiers of the Madras army thirty-two died of cholera in cantonment, and eighty-six when marching, to an average of 10,000 strength; the number attacked being respectively 85 and 200 in 10,000. Dr. Lorimer's reports show that the men were more frequently attacked on long than on short marches, the men (as Dr. Farr observes) being longer exposed to the causes of disease. These causes are those which are incidental to the life of a soldier on the march, such as lying by the banks of rivers, on low marshes, jungly grounds, sleeping on the ground, and encamping amongst the filth of encampments recently occupied, but abandoned—of which indiscretion there were many melancholy examples during the war with Russia in 1854; for example, the occupation of the evacuated camping ground at Aladyn in Bulgaria, and that on the heights above Alma, previously occupied by the Russians, the consequences of which were so fatal to the first and fourth divisions of our army.

The effects of a poor diet in predisposing to cholera will perhaps be better understood by stating that the European suffers less than the Mohammedan, and the Mohammedan, who is better fed and better clothed, than the Hindoo, except during their rigid fasts, when the Mohammedans suffer in a much larger ratio. During the epidemic of 1848 and 1849, in Edinburgh, Dr. William Robertson of that city found that anæmic persons were those most predisposed to cholera.

**Prognosis.**—The mortality from cholera in all countries is very great. Taking the whole number attacked, it is said that the number of deaths in Astrakan were as one to three; in that of Mishni Novogorod as one to two; in Moscow and Kasan as three to five; and in Penza, in the country of the Don Cossacks, as two to three. In the summer of 1831 the mortality at Riga, St. Petersburg, Mitau, Limburg, and Brody, according to the *Berlin Gazette*, was about one-half, while at Dantzic, Elbing, and Posen, it was about two-thirds of the whole number attacked. The period of the epidemic, how-

---

\* The Madras Sepoy, of whom alone Dr. Balfour wrote, invariably carries his family with him. At the end of a long march he puts off his accoutrements, and hastens back, without tasting food, to assist his family out of the difficulties incident to a country in which the roads are often mere tracks. He thus often performs nearly double the route march, and finally encamps on ground which for years has been used for the purpose, and is saturated with the excretion of former sufferers from the disease. Moreover, for a long time the authorities in Southern India were most reckless in sending regiment after regiment in one another's footsteps, through districts known to be infected; and as they all occupied the same encamping ground, the last regiments pitched in places saturated with cholera evacuations, and surrounded by the half-buried remains of the dead. These facts to some extent explain the effects of marching on Sepoys (W. C. MACLEAN).





leading directly to uræmic poisoning, that most practitioners either abandoned its use, or limited it to a mere fractional dose of that usually given in India—namely, from *three* to *twelve* minims of the tincture of opium, or half a grain to a grain of solid opium every four or six hours.

In considering the treatment of cholera there are three periods to be provided for,—(1.) The period of diarrhœa which so frequently precedes cholera; (2.) The algide period, or collapse; and, (3.) Period of reaction.

(1.) *The Period of Diarrhœa.*—To check or arrest the diarrhœa is the practical result aimed at by a variety of formula. Those in which opium is the main remedy have acquired the most amount of confidence.

The management of a case embraces the following points,—(1.) The horizontal position of the body [with perfect rest]; (2.) The administration of opium, with or without cordial stimulants; (3.) The induction of perspiration.

The necessity for the horizontal posture of the patient is, that it aids the efforts of the circulative powers, which tend to weakness.

With regard to opium, its dose must be regulated by (1.) The extent of the nervous prostration; (2.) The rapidity of the dejections; (3.) The extent of vascular depletion. In the cases which present these phenomena in the extreme a much larger dose of opium is required to be given at one time than in the cases less urgent at the outset.

The following formula for pills, each containing a grain of opium, with stimulants, is well known as an anti-spasmodic pill in the early stage of bowel relaxation:

R. Pulv. Opii, gr. xij; Camphor, gr. xxx; Pulv. Capsici, gr. ix; Spt. vin. rect., q. s; Conserv. Rosar, q. s.; Misce et divide in pil xij.

Moderate doses of opium or morphia, either alone or combined with stimulants, as the *pulvis cretæ aromaticus cum opio*, were often sufficient to check diarrhœa. The following (*cholera mixture*, as it was called) was proposed by the Board of Health during the prevalence of cholera, and was no doubt useful in many cases of diarrhœa:

R. Pulveris Aromat., ʒij; Tinct. Catechu, fʒx; Tinct. Cardam. Comp., fʒvi; Tinct. Opii, ʒj; Mist. Cretæ preparat. ad fʒxx. Of this mixture the dose is one ounce.

Bulky doses of remedies are, however, very obviously objectionable; and the usual remedies known as “astringents” (compared with each other, or with opium) have no decided influence for good. “Astringents,” as such, have merely a negative effect.

[The prodromic diarrhœa is said to be promptly arrested by the *lavage Caillard*, composed of 19 parts of the sulphate of soda, and one part of common salt in a suitable quantity of water. *Sulphurous acid* has



with them, or easy of access. During the existence of the epidemic, one person in each factory, &c., should take the charge of the health of the inmates, and should act as 'house physician,' warning all under his or her care to attend to the slightest relaxation of the bowels. He should remind them that the less pain the more danger, and therefore the more need of immediate and energetic action. He should, if possible, ask every individual as to the state of his bowels two or three times a day.

"6. Should the slightest diarrhœa occur, the individual so attacked should at once receive forty minims of solution of morphia or laudanum.\* If from home or at business, he should be at once conveyed home in a cab, put to bed, and kept warm. If chilled, warm water bottles may be put to the feet. If the first dose has not checked the looseness, the patient should take a second, and then have a flannel cloth thoroughly dipped in turpentine, placed all over the stomach and bowels for from forty minutes to an hour, or a large, soft, warm poultice of linseed meal and mustard for one or two hours. If the second dose has not effectually checked the diarrhœa, and medical assistance has not arrived, a third dose may be taken.

"7. The patient must remain in bed two or three days after the diarrhœa is checked. I *insist strongly on this*, for the patient often feels so well that it is difficult to get him to attend to it.

"8. To relieve the thirst, a piece of ice may be given, or a mouthful of iced water, or soda water, but in no case must more fluid be taken at a time, and all food should be abstained from till from fifteen to eighteen hours after the opiate has been administered. *Then*, and for two or three days, the diet should consist of such food as rice, sago, arrow-root, Indian corn flour, tea and toast, &c.; about the third day beef-tea or chicken-soup might be taken.

"9. These rules are for the *first stage, and for it only—i. e., the diarrhœa*. If a person has neglected the first warning, and is in the second stage—*i. e.,* has cramps, vomiting, and stools like rice-water, without smell—you should, till medical assistance arrives, place the patient in bed surrounded with bottles of hot water, and give him a little ice, and mouthfuls of soda and water. If the cramps are severe, you must rub the limbs with turpentine, or chloroform and oil" (*Glasgow Med. Journal*, 1866).

But there are certain cases in which, although the diarrhœa may be altogether checked by such remedies, yet the disease is not cured. Symptoms characteristic of the *algide* stage and collapse supervene. These are the cases which give support to that method of treatment which has for its object elimination by the promotion of purging and of vomiting—excretion of the poison by the alimentary canal. In support also of this method of treatment, its advocates lay stress upon the fact that those are the worst cases in which the diarrhœa is the least; and that those cases are most hopeful in which diarrhœa and vomiting are the most severe.

Dr. Johnson's treatment by castor oil has for its object the elimination of a poison; and it may be said of it, at the outset, that it

---

\* If a measure is not at hand, a small teaspoonful. Of course this dose is for adults. Below that age the doses should be a drop for each year, till twelve or fifteen, and after fifteen a drop and a half for each year, up to forty minims, or a small teaspoonful. More portable than laudanum, and of equal efficacy, would be pills composed of a grain and a half of opium and a grain of cayenne pepper in each pill, three of which may be taken with safety, till medical assistance arrives.



cing vomiting. The pills should be ordered after each discharge from the bowels; but people will find it easier to provide themselves with laudanum than to use complicated mixtures or pills."

[Dr. Leclerc, of Tours, France, and Dr. A. Rodrigues Barraut, of Mauritius, claim to have had large success with the following treatment. The extract of belladonna is given every half hour in quarter-grain doses, and continued until its physiological effects are produced, then increase the intervals to every 1st, 2d, 3d, and 4th hour, giving the remedy until the urinary secretion reappears. Atropia, used hypodermically, was found to relieve the cramps. White of eggs well diluted with water was freely given as a drink. Dr. Hodgen, of St. Louis, who has adopted the theory, and followed the treatment of Dr. Leclerc and Barraut, speaks well of the results. It has also been tried by Dr. J. W. Brewer, U. S. A., and he records a favorable experience.]

In the second or algide stage the object is to promote reaction and to keep it in moderation. If the patient is not seen till profuse discharges—rice-water-like—have taken place, the time for all active treatment has passed, and efforts must be directed solely to restoration and repair.

To promote reaction in cholera and diarrhœa, the following formula has met with most universal approval in this country and in India. So highly is it valued, indeed, that it is ordered to be always in store, and in readiness in the "*Medical Field Companion*" of the army when on the march:\*

R. Ol. Anisi., Ol. Cajeput, Ol. Juniper, āā ʒss.; Æther., ʒss.; Liquor Acid. Halleri, ʒss.; † Tinct. Cinnam., ʒij; misce. *The dose of this mixture is ten drops every quarter of an hour in a tablespoonful of water. An opiate may be given with the first and second dose, but should not be continued, for reasons already given.*

Some physicians think calomel should be given in moderate doses, for the purpose of producing a flow of bile into the intestines, as well as of restoring the other suppressed secretions. The indications, however, more generally followed are to treat the case as we should a similar state in typhoid fever (and calomel in small doses has been shown to be of service in connection with the affection of Peyer's glands in that disease), and to moderate the affections of the bowels by mild opiates, by enemata, and by sinapisms to the abdomen; also, to relieve the head by leeches and cold lotions, and subsequently, as the tongue becomes brown, to support the patient with wine, sago, strong broths, and a generally cordial treatment.

[There is much evidence in favor of the administration of calomel in large doses (20 grains), frequently repeated, to control the vomiting and purging.]

\* A memorandum from Savory and Moore, of date 7th June, 1866, shows that the quantities of the *essential oils* in the mixture now issued are increased to ʒiiss.

† The *Liquor* or *Elixir Halleri* consists of one part of concentrated *Sulphuric Acid* to three parts of *Rectified Spirit*. It is commonly employed in Germany in the treatment of typhus and allied diseases, in doses of five to twenty drops in solution (MURCHISON, *l. c.*, p. 266).



been too freely used in the treatment. In men of intemperate habits we often see, during the stage of reaction, obstinate vomiting of thick, tenacious, green paint-looking matter, probably bile-pigment, acted on by some acid in the stomach or alimentary canal. It is a symptom of evil omen, and often goes on uncontrolled until the patient dies exhausted, and this although all other symptoms may promise a favorable issue. It may last for a week, resisting all remedies, and proving fatal when the urinary secretion has been restored, and all cerebral symptoms have subsided. Alkalies in the effervescing form, free stimulation of the surface, and chloroform in small doses, offer the best hope of relief for such cases. The patient should be nourished more by the bowel than the stomach when vomiting is present.

Ice should be given *ad libitum*, where it can be obtained, not only to dissolve in the mouth, but to swallow in pieces of convenient size (MACLEAN).

A plan, peculiar perhaps to this country, and which was practised to bring about reaction when the inefficiency of medicines was generally admitted, was an injection into the veins of the suffering patient of a solution of half an ounce of *muriate of soda*, and of four scruples of *sesquicarbonate of soda*, in ten pints of water, of a temperature varying from 105° to 120° Fahr. This solution was injected slowly, half an hour being spent in the gradual introduction of the ten pints, and the immediate effects of this treatment were very striking. The good effects were rapid in proportion to the heat of the solution, but a higher temperature than what is stated could not be borne. After the introduction of a few ounces, the pulse, which had ceased to be felt at the wrist, became perceptible, and the heat of the body returned. By the time three or four pints had been injected, the pulse was good, the cramps had ceased, the body, that could not be heated, had become warm, and instead of a cold exudation on the surface, there was a general moisture; the voice, before hoarse and almost extinct, was now natural; the hollowness of the eye, the shrunken state of the features, the leaden hue of the face and body, had disappeared; the expression had become animated, the mind cheerful, the restlessness and uneasy feelings had vanished; the vertigo and noises of the ear, the sense of oppression at the *præcordia*, had given way to comfortable feelings; the thirst, however urgent before the operation, was assuaged, and the secretion of urine restored, though by no means constantly so. But these promising appearances were not lasting; the vomiting continued, the evacuations became even more profuse, showing that the remedy did not touch the root of the evil. Perhaps, if Hallier's observations are correct, it supplied a pabulum for the fostering and development of the mischief. The patient soon relapsed into his former state, from which he might again be roused by a repetition of the injection; but the amendment was transient, and the fatal period not long deferred. Of 156 patients thus treated at Drummond Street Hospital, Edinburgh, under the direction of Dr. Mackintosh, only twenty-five recovered,—a lamentably small





the *salts of zinc*. Each has its advantages, and all may be used. The *carbolic acid*, from its liquid form and from its volatility, is excellently adapted to purify air, and to be used when surfaces are to be washed. It is also useful for sewers and closets. The *sulphate of iron* in substance and strong solution is better adapted for being put in the utensils in a room, as it has no smell, but it may be equally used for sewers and for watering streets. The *sulphate of zinc* (for the *chloride* is too dear) is better adapted for being put on linen or on floors, as it does not iron-mould the linen like the *sulphate of iron*.

"The *carbolic acid* has not been used much in Germany, as it is still too dear; but Pettenkofer makes an observation of importance—viz., that when added to *sulphate of iron* the mixture seems to have more power of preventing ammoniacal development than either substance separately. If so, it might be desirable, as a matter of practice, to use the two together as much as possible. The salts of zinc (*sulphate* or *chloride*) may be also used, but are perhaps not so good, and in some forms are dearer than the iron salts. *Chloride of lime* does not prevent the ammoniacal change, and appears altogether less useful.

"The quantity in which these substances must be used is as follows: For each healthy person, daily, about three-quarters of an ounce of *sulphate of iron*, or one drachm of strong (but impure) *carbolic acid*, are sufficient. This amount will entirely prevent any decomposition of the *æces* for several days. In a town, therefore, where sewers are used, the above amount of *sulphate of iron* or *carbolic acid*, multiplied by the number of persons, should go into the sewers daily, and, if possible, should be passed in from the houses, so as to act on the house drains as well as on the main sewers. If the place is not sewered, then the disinfectants should be added to the cess-pools, middens, latrines, or whatever plans may be in use. If both *sulphate of iron* and *carbolic acid* are used, which is to be recommended, half the quantity of each should be employed. The iron should be dissolved in a good deal of water.

"Dr. Kühne, who has made a great number of experiments on the action of various agents on fermenting substances, does not reckon the value of the *sulphate of iron* or of *carbolic acid* so highly as other observers. He states that neither arrest the various fermentations. Such an arrest, is, however, attained with strong alkalies and strong acids; with *chlorine*, *chloride of lime*, *bromine*, *permanganate of potassium* and *sodium*, and *permanganic acid*. On the hypothesis, therefore (for it is nothing more), that the dangerous condition of the cholera discharges is one of 'fermentation,' he recommends any one of these substances rather than *carbolic acid*, and for common use prefers *permanganate of sodium*, to which (as a concession to Pettenkofer) he adds some *sulphate of iron*. The proportions are two parts of *permanganate of sodium* (solution?), forty-five parts of *acid sulphate of iron*, and fifty-three parts of water in one hundred parts.

"It must be remembered, however, that such points as these must be decided by actual experience, and that arguments derived from the action of these substances on common ferments are not very satisfactory as regards the prevention of cholera.

"In Southampton, in 1866, carbolic acid was chiefly used; and the average amount was about twenty gallons daily for a town of 50,000 people: it certainly appeared useful.

"If an aerial disinfectant is needed, *sulphurous acid* (obtained by burning sulphur) is perhaps the best. *Nitrous acid fumes* are certainly



**History.**—The term *cholera* has been in use since Hippocrates (*Epidem.*, lib. v). Celsus derives it from *χολή*, *bile*, and *ρέω*, *I flow*,—a *bile-flux*; others from *χολὰς*, *intestine*, and *ρέω*—*intestinal-flux*; whilst Trallian and Ruysch give as its derivation *χολήρα*, *the rain-gutter of a house*. Galen gives its true pathogeny. Celsus accurately describes it (lib. iv, cap. 2), and mentions, as an occasional symptom, watery and white discharges; he speaks of it as a dangerous disorder, and one that may quickly cause death. Aretæus (lib. ii, cap. 5) is minute in his account of the gastro-intestinal evacuations. Of modern writers Sydenham has given a most graphic delineation in his narration of the disorder as it prevailed in England in 1669.

**Nature and Pathogeny.**—Cholera morbus is a disorder of hot climates, and the hot seasons of temperate climates. Sydenham says, “It seems partial to a particular part of the year. It sets in at the end of the summer and beginning of autumn, as truly as the swallow comes in spring, or the cuckoo sings in summer.”\* It is most common in that part of the hot season in temperate countries when the temperature during the day is high and falls at night-time; or after those sudden weather-changes so frequent in our summers. Whatever part climate and temperature may have in its causation, it is only collateral and predisposing, the immediate cause in most cases met with in practice being the presence in the stomach and upper bowel of some article of diet, imperfectly digested, which sets up a fermentative and putrefactive process, making the matters acid and acrid, and causing great irritation of the gastro-duodenal lining membrane, which brings on vomiting and purging.† The irritation extends to the liver, inciting, in some cases, the biliary secretion, for a while,—to the spinal cord, causing cramps in the muscles of the abdomen and legs,—to the sympathetic, producing, through the periphero-vasal system, coldness of the skin and capillary torpor. The most common offending articles of diet are shell-fish, salted or tainted meat, and decaying vegetables. From the peculiar state of the digestive organs, sound food may not be acted on by the gastric juices, and, undergoing the putrefactive process in the stomach, becomes an exciting cause. Drinking largely of iced-water, when the body is overheated, is an occasional cause. Emanations from cesspools and sewers, and putrilage, have produced a train of symptoms identical with those of cholera morbus.

Cholera morbus is a disorder essentially distinct in its nature and ætic genesis from Asiatic cholera. The one is caused by a material toxic agent directly in contact with the gastro-intestinal membrane, rendered specially susceptible to its action from the influence of season and temperature; the other owns for its cause a specific poison, infecting through the atmosphere and the evacuations of those suffering from it. The discharges of cholera morbus are acrid, dark-colored, and, often, during some time in an attack, bilious; while in Asiatic cholera, there is no evidence of the presence of bile in the evacuations, and they are light-colored, and turbid from whitish particles—the epithelial cells of the mucous membrane of the stomach and bowels.

**Symptoms.**—An attack of cholera morbus is most often sudden,—though it may be preceded by nausea, colicky pains, and rumbling in the belly—beginning with incessant, uncontrollable simultaneous vomiting and purging; first of the usual contents of the stomach and bowels, and

---

\* *Med. Obser.*, ch. ii. Works of T. Sydenham. Syd. Soc. ed., vol. i, p. 168.

† Galen attributes cholera morbus to the presence of acrid humors generated by the corruption of the food.



A chief point is to withhold all drink, but small pieces of ice may be held in the mouth, the water being spit out. Dry rubbing with the hand, or with woollen cloths, should be made over the body, and particularly over the abdomen and lower extremities, to relieve the cramps, and revive the capillary circulation. Sinapisms may be applied to the calves of the legs, inside of the thighs, and along the spine, in severer cases; or the upper and lower extremities, belly, chest, and spine, be covered with woollen cloths, well wrung out of hot water, to which mustard flour has been added, and the patient then wrapped in a dry warm blanket. If prostration is very great with a tendency to collapse, a mixture of chloroform, camphor, ether, and capsicum, will prove an excellent stimulant, and has sometimes quickly checked all the symptoms. Iced brandy and iced champagne are favorite stimulants in an advanced stage of the disorder. On the abatement of the acute symptoms, the tongue remaining furred, a mild mercurial, followed by a small dose of castor oil, may be administered without risk of bringing on a relapse.]

[CHOLERA INFANTUM—*Summer Complaint*—*Infantile Cholera*.

(DR. CLYMER.)

**Definition.**—*A perilous disorder of early infancy, most common in the first year of life, the chief symptoms being stubborn purging of variously colored serous fluid, and vomiting; occurring under the combined influence of high atmospheric temperature, great humidity, and malnutrition. The solitary follicles of the intestines are more or less diseased.*

**History.**—Cholera infantum has been thought by many writers to be a disorder peculiar to the United States, but, as Dr. Trousseau remarks, it has always been observed, and shows itself in all countries under the same circumstances of season, age, and bodily state, and he describes it as *cholera infantile* or *mal d'été*.\* Sydenham says, "There is a sort of cholera morbus exceedingly fatal to infants."† It is probably more generally prevalent in the Middle and Western States than in Europe, recurring with uniform seasonal regularity, and being the chief cause of the great loss of life amongst infants in the large towns of those States during the summer months. There is no doubt that in the death-records distinct pathogenetic affections are ignorantly reported under the same general head of cholera infantum,—as the diarrhœa of dentition, cholera morbus, enterorrhœa, enteritis, and typhoid fever; yet we must admit the existence of a disorder of early infancy, of a special nature, characterized by typical symptoms, bred by the operation of the same causal factors, and whose true pathogeny and precise phenomena have yet to be accurately studied and described.

**Symptoms.**—One form of cholera infantum resembles very much the cholera morbus of adults; is sudden in its invasion; and has for its chief symptoms incessant and violent purging and vomiting of serous fluid, of a greenish or yellowish hue; great thirst; sharp and incessant cries, betokening pain; cold surface; quick pulse; and early collapse. Such cases may end fatally within twenty-fours. The more chronic and common form of the disorder begins with looseness of the bowels, soon followed by steady vomiting. The intestinal evacuations are at first fecal, very liquid, sour and offensive, but soon become serous, of a light-yellow color—like water to which a little yolk of egg has been added, or

\* Clinique Médicale, 2d ed., t. 3, p. 128. Paris, 1865.

† Works of Thomas Sydenham, M.D.: Processus Integri, ch. xxiv, vol. ii, p. 267; Syd. Soc. ed., London, 1850.



eleven years, out of 1245 deaths from cholera infantum, 1061 were in the months of July, August, and September. During the first twenty-eight days of July, 1866—a month of unusually great heat—there were reported 687 deaths from cholera infantum, in a total mortality from all diseases of 3452. In the preceding trimester, in a total mortality of 5597, the deaths from cholera infantum were 76. The influence of *high temperature* is more striking, if the weekly mortality of the month of July, 1866, from cholera infantum, is examined. For the week ending July 7, mean temperature  $81\frac{3}{4}^{\circ}$  Fahr.: total deaths, 493; deaths from cholera infantum, 61. For the week ending July 14, mean temperature  $82\frac{1}{4}^{\circ}$  Fahr.: total deaths, 827; deaths from cholera infantum, 172. For the week ending July 21, mean temperature,  $82\frac{5}{7}^{\circ}$  Fahr.: total deaths, 1362; deaths from cholera infantum, 278. For the week ending July 28, mean temperature,  $73\frac{1}{2}^{\circ}$  Fahr.: total deaths, 770; deaths from cholera infantum, 176.

The late Dr. James Stewart instituted a series of observations to ascertain the relation of *atmospheric humidity* to cholera infantum, and came to the conclusion that ordinary climatic humidity had but little influence in its development; but, directing his attention to the occasional state of the dew-point, as it occurred in localities where the disorder was most rife—the population living in overcrowded underground cellars and tenement-houses—he “discovered a great difference within doors between it and the general dew-point of the external air, continuing often for a long time.” His observations showed—(1.) That the moisture was always greater nearer the surface of the earth, the difference at times being  $4^{\circ}$ ; (2.) That in very hot weather, in crowded rooms at night, “when all were within, the dew-point is very nearly the temperature of the air, thus saturating it with moisture.” With a temperature of  $90^{\circ}$  to  $95^{\circ}$ , and a dew-point in a crowded room almost equal to the temperature, a feeling of suffocation is experienced, which is easily accounted for when it is known that the dew-point of the breath, as it is expelled from the lungs, is  $94^{\circ}$ , and that the mean dew-point of the atmosphere is  $38^{\circ}$ ; and also, that in the hottest weather it rarely exceeds  $70^{\circ}$ .\*

*Malnutrition* is due to several sources. The depressing and exhausting effects of great heat and moisture are exerted on a body imperfectly nourished. The mother, suffering from the devitalizing and septic influences which surround her, secretes a milk imperfectly elaborated and unfitted for food; or, when in itself wholesome, the digestive organs of the infant are so disordered, that there is a lack of the changes it should undergo; a chemical act of decomposition happens, directly the opposite of the vital act of digestion, and cheesy clots of the coagulated casein are rejected in the vomit, or pass unaltered or corrupt through the bowels. Infants brought up by hand, as well as those just weaned, are very liable to cholera infantum. Here a deleterious and inadequate diet—often exclusively farinaceous or gelatinous, or of milk from diseased animals, or so dilute as to be unfit for food—plays a chief part. The direct influence of *animal poison* must not be passed over. The chief haunts of cholera infantum are the fever-nests and cholera-fields of large towns. Diphtheria, scarlet fever, putrid sore throat, typhus, and Asiatic cholera, herd together, and fester in the slums, where the wretched and squalid dwellers are stowed away in ill-ventilated, ill-drained underground cellars and tenement-houses, in the midst of every possible insanitary condition; breathing a septic atmosphere, poisoned without, by the putrilage of slaughter-houses, soap and glue factories, cesspools and sewers, and

---

\* Loc. cit., p. 290.





which laudanum is poured, applied to the spine, will be found useful in checking vomiting. The skin, in common with all the excreting organs, is inactive. The effect of great and prolonged heat is always to increase its function; there is for awhile local hyperæmia, with swelling of the papillæ, giving rise to the eruption familiarly known as prickly-heat. Finally there is loss of tone and sluggishness, especially when exposed to currents of air; and its function must be excited. This may be done by gentle friction by woollen cloths, or a warm alkaline bath, in which the little patient should not remain longer than three minutes, and then be quickly dried, and wrapped in flannel. So soon as the stomach and bowels will tolerate food, it should be given of fitting quality, and in proper quantity. Farinaceous food (the carbo-hydrates),—the usual diet in all diarrhœal disorders,—is entirely inappropriate. The starches are unaltered by the peptic solvent, and turn acid in a mass; very often in the mouth, from the action of the saliva, so abundant in infants, before they reach the stomach. In infants so fed they will be found in the bowels almost unchanged (N. GUILLOT). The natural food of the infant is entirely dissimilar from these amylaceous articles, which not being digested and assimilated, are consequently not only innutritive, but act as direct irritants to the alimentary mucous membrane. Limed milk, to which a little gelatine has been added, or rennet whey, may be given; but in protracted cases, where the prostration is great, and emaciation rapid, beef essence, freshly prepared and well salted, will often be well borne and quickly appropriated. The avidity with which the little patient takes animal food\*—seizing on meat, salt fish, &c., when it can,—shows an instinctive craving for proper nourishment, too constantly withheld from false notions of the nature of the disorder. Some twenty-five years since, Dr. Weisse, a Russian physician, used raw meat in a case of infantile chronic diarrhœa, and with success. It is a popular remedy in some parts of Europe in chronic and wasting affections. His example was followed by others, especially by Dr. Trousseau, of Paris.† Lean beef or mutton is first finely hashed, pounded in a mortar to a pulp, and then passed through a fine sieve; a thick concentrated juice—*purée de viande*—is thus obtained, nutritious and digestible, and often, when salted or otherwise flavored, quite acceptable. Give a half to three-quarters of an ounce, in fractional doses, the first day; and if well borne by the stomach, increase the quantity day by day, until a quarter or half a pound is taken in the course of the twenty-four hours. Dr. Trousseau mentions a case in his own family, where he continued it for more than one year; the child, about two years of age, finally taking a pound of raw meat daily. For the first day or two much of it may pass hardly changed in the stools, but this alone should not prevent it being persevered in. From the observations of Weisse, Braun, Van Siebold, and Trousseau, this diet, if long continued, is liable to generate tænia. White of eggs, thinned with natural or artificial Selters, or Vichy, or weak lime, water, is an excellent drink; or a few grains of bicarbonate of soda, may be added to the albuminate. Tonics and stimulants are very frequently required in the course of the disorder. Of the former, minute doses of arsenic (the liquor arsenici chloridi), alone or combined with quinine, or

---

\* ["I have seen many children recover," said the late Dr. Rush, in reference to the treatment of cholera infantum, "from being gratified in an inclination to eat salted fish and meat. In some instances they evince an appetite for butter, and the richest gravies of roasted meat, and eat them with obvious relief to all their symptoms."—*Medical Inquiries and Observations*, 2d ed.

† Clinique Médicale, par A. Trousseau, t. iii<sup>e</sup>, 2<sup>e</sup> ed., Paris, 1865.]



than a minute, by itching in the bitten part, and very soon a wheal, or circumscribed pale swelling, with a nearly level surface and a circumscribed border, gradually rises and extends in the skin. The swelling is produced by œdema of a small portion of the cutis at and around the bite. As the itching subsides, the pale swelling becomes less defined, and the more general vascular swelling of the surrounding and adjacent tissues gradually encroaches on the primary swelling at the bitten spot. In about twenty-four hours a papule or some form of secondary inflammation appears, with renewed itching at the site of the puncture. This, too, in the case of the bug-bite, gradually subsides. The primary swelling here described illustrates the immediate effects of the morbid poison on the tissue at and round the seat of inoculation, and within the area of such a swelling the tissues are, by the direct contact or influence of the venom, altered in their nutritive relation to the blood. Such specific alterations of the tissues at the seat of inoculation occurs with the syphilitic, the vaccine, and such-like virus; but the direct influence is most rapidly shown in the effects of the bites of the viper, the rattlesnake, and the *cobra di capello*. In such cases sloughing of the areolar tissue is established immediately after the bite. The poison seems to operate at once on the tissue, neither in the direction of the nerves, nor of the absorbents, nor of the bloodvessels; but the slough forms at the puncture, as if the venom had completely and at once killed the tissue (BRODIE, PAGET).

A secondary inflammation soon appears at the bitten or punctured part; and the occurrence of this new inflammation may be ascribed, in some measure, to an influence exercised by the virus on the blood; and it proves that the part does not return to health, although the first effects of the inoculation may subside. It proves that some material of the virus remains, or that the effects it has already produced upon the tissues at the injured part alter their relations to the blood, and render the part prone to specific disease. These specific effects upon the part may remain locally quiescent for a considerable length of time—during all that period of latency or incubation which intervenes between the inoculation and the appearance of the specific disease. But during all this interval—during all this period of incubation—the tissues at the site of inoculation are constantly changing; and the virus itself, like all organic matter, is probably in constant process of transformation till the *zymosis* is complete, and the specific disease is fully developed and expressed by various constitutional phenomena.

Dr. George B. Halford, Professor of Anatomy in the University of Melbourne, has recently (*Brit. Med. Journal*, July 20, 1867) given an interesting account of the action of the poison of the *cobra* upon the blood.

“When a person is mortally bitten by the *cobra di capello*, molecules of living ‘germinal’ matter are thrown into the blood, and speedily grow into cells, and as rapidly multiply, so that in a few hours millions upon millions are produced at the expense . . . of the oxygen absorbed into the blood during respiration; hence the gradual decrease and ultimate ex-



the ordinary examination of the blood goes, the post-mortem appearances are similar to those seen after drowning and suffocation."

The changes which the absorbed virus undergoes in the living and infected body are,—(1.) Increase; (2.) Transformation; (3.) Combination; and (4.) Separation or excretion.

The *increase* of the virus is shown in such inoculable diseases as *vaccinia*, *glanders*, *malignant pustule*, *syphilis*. In all of these diseases the inoculation of the minutest portion of virus is followed by the formation of one or more vesicular structures, containing fluid from which virus, similarly and equally potent, is produced in million-fold quantity. Thus the virus of any contagious disease developed in an infected person may render his exhalations capable of similarly affecting thousands of other people. And it is probably among azotized materials chiefly that morbid poisons, whether of animal origin or of disease, find the means of their increase (CARPENTER, PAGET, SIMON).

The *transformation* of the virus is indicated by the successive phenomena which supervene during the continuous course of a specific disease. For example, syphilis is followed by a series of secondary and tertiary phenomena, which follow, on the whole, a uniform course in a great variety of patients; so that these regular syphilitic phenomena may be attributed to the *transformations* of the morbid poison; while the irregularities of the phenomena may be ascribed to constitutional peculiarities of the patient, either natural or acquired from treatment. Thus there are periods of incubation, of development, of maturity, and of degeneration in the material of the virus; and the various phenomena which constitute the *symptoms* and *prodromata* of the disease correspond to such periods of transformation; while the increasing disturbance of the general health probably implies that the morbid poison is increasing while it is being transformed—that it grows or multiplies with its development.

The *combination* of a morbid poison with some normal material of the blood is indicated by the circumstance, that when the same specific disease, produced by the inoculation of the same matter, affects many persons, the disease set up in each of them may present different peculiar features. The disease may have some peculiar and varied methods of expressing its development in different persons—"personal peculiarities," as Mr. Paget calls them, and which he considers due in some measure to the combination of the virus with one or more of those normal materials of the blood which have in each person a peculiar or personal character. By such combinations the following characters of specific diseases may be explained, namely,—(1.) Changes in the disease by transmission from one person to another; (2.) Some varieties of syphilitic sores, and varieties of their consequences in different persons inoculated from the same source; (3.) The change in the forms of secondary syphilis in transmission from parent to offspring.

The *separation* or *excretion* of the virus may be accomplished in many different ways, and may be regarded as the final purpose of



## CHAPTER VII.

## DETAILED DESCRIPTION OF THE ENTHETIC ORDER OF ZYMOTIC DISEASES.

## HYDROPHOBIA.

LATIN Eq., *Rabies*; FRENCH Eq., *Hydrophobie*; GERMAN Eq., *Wasserscheu*; ITALIAN Eq., *Idrofobia*.

**Definition.**—*A disease peculiar to animals of the canine or feline race, the specific poison of which, being implanted by them in man, or in other animals, produces a similar malady. The saliva or secretion issuing from the mouth of the diseased or rabid animal conveys the poison which inoculates rabies, either through a wound or through a thin epidermis without abrasion. The period of incubation of the poison after inoculation varies from four to sixteen weeks, or even longer, before the malady becomes developed. The disease is characterized by severe constriction about the throat, spasmodic action of the diaphragm, and distress at the epigastrium: all of which are aggravated or brought about by attempts to take fluid, or by the least breath or current of air on the surface of the body, which produces, in the first instance, an effect resembling that produced upon stepping into a cold bath. Tenacious and clammy saliva issues from the mouth. Paroxysms of phrensy, or of uncontrollable impulsive violence (rabidity), supervene. The duration of the disease varies from three to six or seven days, the greater number of cases terminating in death on the second and fourth days from the accession of symptoms. Death is generally sudden, and unexpected at the moment.*

**Pathology and Symptoms.**—The saliva of the dog or other animals laboring under rabies is either the virus, or contains (as any menstruum would) the poisonous principle which by inoculation produces *hydrophobia* in the human body. The disease is so named, not because there is any dread of water, but because in man the most prominent symptom is an inability to swallow, or to attempt to swallow, any fluid, on account of the extreme spasms which the attempt produces. The experiments of Hartwig have proved that the poison is of a definite character, that it may impregnate various substances, and that it retains its activity for a long period.

Two points in the pathology of *rabies* are peculiar—namely, *first*, that a long period of latency exists in the human subject; and, *second*, that inoculation is not always followed by the development of the specific disease.

With regard to the first of these peculiarities, it is to be noticed that, although in some cases pain has been felt in the cicatrix a considerable time after the accident, and in a few a slight fever or a rapid pulse has been remarked to continue from the receipt of the





Thomson in vol. i of the *Lancet*. The subject of it, a lad aged eighteen, had been twenty-five months in close confinement in prison, and during that time had never been exposed to the bite of any animal. He had been bitten severely by a dog seven years before in the right hip, and a scar still remained. During the whole period he was under observation he was sullen, gloomy, and reserved, and was never known to look the person in the face to whom he spoke. Death occurred after a three days' illness, during which "the most decided symptoms of hydrophobia were manifested." On the 15th of May, 1854, a case was admitted into Guy's Hospital, under the care of Dr. Hughes, in which hydrophobia appeared to have been developed five years after the bite (*Med. Times*, 1854).

Such observations render it extremely probable that the period of incubation of the specific poison of hydrophobia is indefinite; and the circumstances which, in man especially, seem to shorten the duration of this period or prolong it, are in a great measure quite unknown. There are some other circumstances which seem to show that during the long interval of apparent latency the quantity or the virulence of the implanted poison seems to increase, locally at least, if not also more extensively in the system.

*First*, In some instances there are evidences of a slow and silent change going on in the constitution, indicated by sallow looks, sunken eyes, a pulse somewhat accelerated, more easily excited and weaker, combined with symptoms of general debility (COPLAND.)

*Second*, The observations of Dr. Marochetti, who visited the Ukraine in 1820, and who maintained that in that country characteristic pustules were observed to form beneath the tongue, near the orifices of the submaxillary glands, between the third and ninth day after the infliction of the bite. This observation was confirmed by M. Magistel, at Boulay, in France, in 1822, who noticed that the pustules formed from the *sixth* till the *thirty-second* day. He observed two forms of pustules, a crystalline and an opaque, the latter of which, when opened, left a small ulcerated cavity. They were situated on the sides of the *frænum linguæ*, and on the lateral parts of the inferior surface of the tongue.

*Third*, Changes which take place in the cicatrix before the development of characteristic symptoms, indicate that the implanted poison there undergoes some process, the nature of which is as yet not known.

After the local incubation of the poison is complete, its specific action appears to be exercised upon the *medulla oblongata* and the *eighth pair of nerves*, and subsequently lesions of the structures supplied by the branches of the *eighth pair*. The action of the poison appears in the first instance to be made distinctly manifest by the œsophageal branch of the *eighth pair*, producing that derangement of function which gives rise to the characteristic symptom of the disease, or to the extreme difficulty of swallowing, especially of fluids; while the spasmodic catching of the breath, consequent even on touching the lips with any liquid, proves that the recurrent nerve is equally affected. Subsequently the eye and ear become distressed by every ray of light or impulse of sound,



congestion. The salivary glands have likewise occasionally been observed increased in size, and vascular. In a case of hydrophobia which I had an opportunity of dissecting at Renfrew, near Glasgow, the most prominent morbid change was visible in the greatly increased vascularity of the lungs, and of the mucous membrane of the back part of the mouth, pharynx, and larynx, as far as the vocal cords. The whole of these parts were covered by a tenacious frothy mucus, tinged with blood. The glands surrounding the papillæ over the back part of the tongue were very much enlarged, not unlike what I have observed in severe cases of cholera. So also were the submucous glands of the pharynx, the epiglottis, and the larynx, even in its cavity, and of those beneath the tongue. Inflammatory appearances in these parts have been observed by Morgagni, Babington, Watt, Portal, Troillet, Copland, and others.

**Symptoms.**—The wound inflicted by the bite, whether neglected or dressed, generally heals up kindly, leaving a cicatrix, and for a time the patient usually suffers no other derangement of health than the depression of spirits which his apprehensions are calculated to excite. A few weeks or a few months having elapsed, the latency of the poison terminates, and the disease is formed. The course of the affection is usually divided into three stages, the first stage comprising the symptoms which precede the difficulty of swallowing; the second commences with the difficulty of swallowing, and terminates with the overthrow of the mind; the last stage embraces all the concluding phenomena.

The first stage commences in a few instances by the patient's attention being roused by a numbness extending toward the sensorium from the injured part (which, if an extremity, may become tremulous); or pain is felt in the cicatrix, sometimes severe and sometimes trifling, and which shoots up the bitten limb, following in general the course of the nerve towards the trunk. It shoots as if towards the heart, but there is no evidence of lymphatic absorption. Pain, however, is by no means constant, and is for the most part absent. In the latter case the first symptom is chilliness, with headache, or a slight attack of fever, and the patient is more excited or depressed than usual. These premonitory warnings last but a few hours, or at most a few days, when the fatal but characteristic symptom, "the difficulty and dread of swallowing"—a symptom which distinguishes this malady from all others—appears, and the hydrophobic stage commences.

The second or hydrophobic stage is ushered in with a great difficulty, if not an utter impossibility, of swallowing any liquid—a symptom which generally comes on suddenly; and such horrible sensations accompany the effort, that whatever afterwards even recalls the idea of a fluid excites violent agitation and aversion. Some patients who have been able to give some account of themselves describe the hydrophobic sensation as a rising of the stomach which obstructs the passage; others as a feeling of suffocation, or a sense of choking, which renders every attempt to pass liquids over the root of the tongue not only impossible, but which excites convulsive action in the muscles of the larynx, pharynx, and abdo-

men. In this state, says Dr. John Hunter, "the patient finds some relief from running or walking, which shows that the lungs are not yet the seat of any great oppression."

The hydrophobia, or inability to swallow fluids, is shortly accompanied by an increased flow of saliva, termed the "hydrophobic slaver." This secretion, as the disease advances, is not only copious but viscid, so that it adheres to the throat, and causes incessant spitting, and the quantity expectorated may be taken as the measure of the violence of the disease. By some this increased flow of saliva is considered as an effort of the system to eliminate the poison through these excretory glands; and therefore, mercury in large doses, to promote salivation, has been recommended to promote elimination in this way, and to reduce the extreme excitability of the nervous system (LIGGET).

The aversion to fluids is no sooner established than another series of symptoms of dreadful severity, or a highly exalted state of every corporeal sense, is added. Indeed, it is hardly possible to depict the sufferings of the patient from this cause; for not only does he shrink at the slightest breath that blows over him, but the passage of a fly, the motion of the bed-curtain, or any attempt to touch him, produces indescribable agony, almost amounting to convulsions. Dr. Elliotson states that the effect produced by these causes very much resembles that produced upon stepping into a cold bath. The sense of sight is no less a source of terror than that of touch, for the approach of a candle, the reflections from a mirror or other polished surface, occasions the same distressing effect. The hearing is as strongly affected as the other senses, so that the least noise, and especially that of pouring out fluids, throws him into a fearful paroxysm. An attendant who sat up with a hydrophobic boy made water within his hearing, which threw the sufferer into a most violent agitation. The degree to which this painful state of the senses arrives may be understood when it is stated that Magendie records the case of a deaf and dumb child who heard distinctly in this stage of the disease. The patient, thus incessantly harassed and pained by every circumstance around him, becomes peevish and irritable, and at length sees his family, relations, and strangers, with feelings of dislike and aversion, and sometimes apparently with horror.

The third stage commences by the cerebral functions becoming disturbed, the mind being either filled with dreadful apprehensions, or being so completely overthrown that paroxysms of uncontrollable impulsive violence follow. A rabid impulse overtakes the patient to tear in pieces who and whatever opposes him. This rabid impulse greatly distresses him; and it is often strongest against those to whom he is most attached, although he struggles to suppress it. In this stage horror is strongly depicted on the countenance: every symptom is aggravated, the saliva grows thick and ropy, while the poor sufferer, not daring to make the slightest attempt to swallow, spits it out incessantly, oftentimes with frequent retchings and vomiting. In this state he sometimes turns black in the face, falling into convulsions, in which he expires; or,

exhausted by his great efforts, a sudden calm ensues, and, as if nature gave up the struggle, he dies without a groan.

**Remote Cause.**—Hydrophobia originates in animals of the canine and feline races, as the dog, the fox, the wolf, the jackall, and the cat, as a specific inoculable disease, but from what peculiar source is altogether undetermined. It is probably at all times to a certain extent endemic, and occasionally epidemic among these animals. It has been supposed that it is excited in them by the great heat of the dog-days, or by the *æstus veneris*; but Troillet has shown that canine madness occurs with nearly equal frequency in winter, spring, summer, and autumn. The poison is not peculiar to any country. Rabies is found equally in Europe, Asia, and America. Neither is it limited to climate. It prevails in the frozen regions of Canada, as well as in the East and West Indies. The difficulties attending any explanation of the origin of this poison are at present not to be surmounted; but hydrophobia once originated in the animals that have been mentioned, they have the power of reproducing it by their bite, not only in each other, but probably in all warm-blooded animals, certainly in all domesticated animals, as the horse, the elephant, the sheep, the ox, even in the common fowl, and in man. It will be necessary to the proper understanding of hydrophobia to give a short outline of the disease as it occurs in the dog, so constantly associated with us in domestic life, and the principal source of the disease in the human subject.

The symptoms of this formidable affection, as witnessed in the dog, are some singular departure from his ordinary habits, such as picking straws or small bits of paper off the floor, and swallowing them; licking the noses of other dogs, or other cold surfaces, such as stones or iron. Besides this, he is observed to be more lonely, shy, and irritable; his voice is so changed that his bark would not be recognized by those who have known his voice before; and he is less eager for his food, or refuses it altogether. His ears and his tail droop; his look is suspicious and haggard; and sometimes from the very commencement, there is a redness and watering of the eyes. In a short time saliva begins to flow from his mouth, he “slavers,” his fauces may be seen to be inflamed, and he is feverish. The animal, though highly irritable and easily provoked, still obeys the voice of his master; and it is remarkable “that the dread of fluids, and even the sight of them—so striking a feature in man—is often wanting in dogs and other animals, for many dogs lap water during the disease” (YOUATT). In many dogs the symptoms never rise higher than these; but in others there is a repugnance to control, and a readiness to be aroused to extreme rage, on the appearance of a stick, whip, or other instrument of punishment, or on any attempt at intimidation, which strikingly characterizes the disease. In this state, however, he seldom fights a determined battle, but bites and runs away; still even this mitigated irascibility usually ends in indiscriminate aggression, till at length he dies, apparently, of convulsions or asthenia, or from mere nervous excitement and functional derangement. Magendie has inspected the hydrophobic dog, and found no characteristic morbid change. In all cases, how-





nill observes that "such of them as have been thought to become affected merely by the contagion of the same kennel will generally be found, upon minute examination, to exhibit the marks of bites, though concealed by the hair." When a scratch or other abrasion exists, a rabid dog merely licking the part is sufficient to implant the poison of rabies.

**Diagnosis.**—When hydrophobia is fully formed there is no disease with which it can be confounded; but there are many reported cases in which the imagination of a patient bitten by a dog has been so powerful as to induce symptoms resembling the disease. In hysteria the difficulty of swallowing exists, but no other symptom. Tetanus is the disease with which rabies is most apt to be confounded; yet the differences are sufficiently marked. The spasm of the muscles is more continued in tetanus; less remitting, and never intermitting. The jaw is usually much in motion in hydrophobia, in frequent attempts to clear the mouth and throat from the peculiar tenacious mucus; in tetanus it is fixed. Tetanus is rarely attended with aversion to liquids; on the contrary, the bath is grateful; nor are the tetanic paroxysms increased by the sight, hearing, or touch of fluids. Also, tetanus makes its accession usually at a much earlier period after infliction of the injury. Physiologically, while tetanus is a disease of the true spinal system, hydrophobia involves the brain also, as evinced by the disorder of intellectual function and special sense, even early in the disease. Further, the two diseases differ greatly in their mode of induction. Tetanus, in the traumatic cases, is caused by irritation of a nerve, and by disease of the spinal marrow in those which are idiopathic. Hydrophobia is the result of a specific poison introduced into the circulation, and thence affecting the nervous system as a poison would (MILLER). While in tetanus the stimulus which excites the paroxysms "operates through the true spinal cord, in hydrophobia it is often conducted from the ganglia of special sense, or even from the brain, so that the sight or sound of fluids, or even the idea of them, occasions, equally with their contact, or with that of a current of air, the most distressing convulsions" (CARPENTER).

**Prognosis.**—There are few instances of any patient or animal suffering from this disease having recovered.

**Treatment.**—As there are but very few authenticated cases of recovery from hydrophobia, so there are few instances of any mitigation of the symptoms by the use of medicine. All that remains is to mention the most leading experiments that have been made, with the hope that, as they have not been successful, they may not be wantonly repeated.

Dr. Hamilton gives twenty-one cases, and adds—"many hundreds more are on record," in which venesection has been unsuccessful, though frequent and copious. Opium has been given by Dr. Babington, to the enormous amount of 180 grains of solid opium in eleven hours, without the slightest narcotic effect, or the slightest mitigation of the symptoms. Nord has given a drachm of belladonna in twelve hours, without any benefit. Dr. Atterly gave to a child eight years old two drachms of calomel by the mouth, and





many, and Italy, upon animals, has been entirely unsuccessful. M. Grindard conceived that the vaccine virus might influence hydrophobia, and he vaccinated a hydrophobic child in three places, and afterwards injected five charges of vaccine lymph into the veins; but the child died without any marked remission, and in the usual time. The following draught has been found rather to promote euthanasia than to hold out any prospect of cure:

R. Spirit. Æther. Sulph., Tinct. Opii, āā ℥xx; Spirit Ammon. Aromat., ℥ss.; Chloroform, ℥xx; Mist. Camph., ℥iss.; *misce.* To be given as often as may be considered safe (CUNNINGHAM, CARDEN).

On the same principle chlorodyne ought to be given. (For its composition, see page 454, *ante.*) The vapor bath is sometimes useful in moderating spasm.

**Preventive Treatment.**—The probabilities are, that unless the operation of excision, or cauterization, be performed within a few minutes after the bite of the rabid animal, it is impossible to save the patient from the fatal disease, which, according to the susceptibility of his constitution, may threaten him at any moment. In all probability no prophylactic medicine exists in nature, and the administration of any potent substance by way of prevention is worse than useless; for, without protecting the patient, it may injure his constitution. Mild remedies, if they tend to tranquillize his mind and appease his apprehensions, may be innocently employed.

The theory which maintains that a zymotic incubation first takes place in the wound, by which the poison is originally implanted, suggests the most rational prophylactic—namely, to destroy entirely by *potassa fusa* the whole cicatrix, where practicable, or by some other surgical means entirely to remove it, at as early a period as possible, and *previous* to the occurrence of symptoms. When premonitory symptoms are first observed, the following plan has the recommendation of Dr. Maxwell in *The Indian Journal of Medical and Physical Science*, and of Dr. Copland, namely,—(1.) That the original cicatrix be freely laid open, and suppuration from it speedily and freely produced. (2.) The nerves, or nerve, leading to the part are to be divided without delay, the more remote from the wound the better. (3.) Free perspiration should be promoted by the hot air bath. (4.) Bleeding from the arm to syncope in robust persons with sthenic symptoms, or cuttings on the nape of the neck, are modes of practice indicated by the lesions found after death.

#### GLANDERS.

LATIN EQ., *Equinia*; FRENCH EQ., *Morve*; GERMAN EQ., *Rotz*; ITALIAN EQ., *Cimurro*.

**Definition.**—A febrile disease of a malignant type, resulting from the implanting of a specific poison from glandered horses. It is characterized by vascular injection of the nasal mucous membrane, from which



*Society*, 1862). The attention of the profession was first called to this interesting subject by Mr. Muscroft, in *The Edinburgh Medical and Surgical Journal*, in the year 1821, where he relates the case of the whipper-in of the Bradworth hunt, who wounded himself in cutting up a glandered horse for the kennel, and died, at the end of a week, of confirmed glanders; and two similar cases appeared in the same work about two years afterwards. Simultaneously with Mr. Muscroft, Dr. Copland, in the course of a discussion at the Medico-Chirurgical Society of London, stated that the fact of the disease having been thus communicated had been proved by cases that had occurred in Germany, and which were published in *Rust's Magazine* for 1821. The cases excited but little notice till Mr. Travers published his valuable work on *Constitutional Irritation*, in 1828, containing a letter from Professor Coleman on the transmission of glanders from the horse to man, and from man to the ass, together with some other cases which had fallen under his own observation. The subject was now followed up by Dr. Elliotson, in two papers in the *Transactions of the Medico-Chirurgical Society*, narrating three cases which had occurred in his own, Dr. Roots's, and Dr. Williams's practice. At length all then known facts were collected in an elaborate paper by Rayer, in the sixth volume of the *Mémoires de l'Académie Royale de Médecine*.

In the cases collected by Rayer, the nose and nasal fossæ had only been examined in four cases out of fifteen, and in these there was found either ecchymosis, ulceration, or gangrene of the mucous membrane of the *septum nasi*, or of the sinuses. The mucous membrane of the larynx, or trachea, has likewise been found studded either with the peculiar eruption, or diffusely inflamed or ulcerated, so much so that in one case the epiglottis was in part destroyed. The lungs have likewise been found either gorged with blood, or the seat of lobular pneumonia, or of vomicæ, with typhoid symptoms—*broncho-pneumo-typhus*, as it is called in Germany. In Dr. Roots's case there was an encysted abscess of the lung, which contained about two ounces of pus. Besides these affections of the more vital organs, a number of small farcy tumors have been found in different parts of the trunk and extremities, and perfectly remote from the point originally punctured. These tumors were in different states of inflammation, some being white and indurated, others soft and injected, and others in a state of suppuration. In Dr. Roots's case an abscess on the back of the hand communicated with the articulation of the metacarpal bones; and in another case an abscess had opened into the knee-joint. The absorbent vessels have likewise been found inflamed along the arm from the point of puncture, or site of primary inoculation, and the glands to which they lead have been found enlarged and indurated, or in a state of suppuration.

The result of all these observations shows that in cases of glanders a specific poison is implanted which infects the blood, and, after a given period of latency, produces, in slight cases, an abscess at the point of puncture, followed by some tumors in the course of the absorbents connected with the punctured part. In severe cases



by inflammation of the lymphatics proceeding from the wounded part, and extending sometimes to the elbow or axilla, and involving the axillary glands. The effects are followed by inflammation and extensive abscesses in the subcutaneous cellular tissue, often involving the whole limb. From this state the patient may recover; but should these abscesses be multiplied over various parts of the body, and be accompanied either by the pustular or gangrenous vesicular eruptions, or by both, the result is generally fatal; hectic symptoms supervene, and hasten the final catastrophe.

The disease has terminated within a fortnight, but more commonly it has not proved fatal till the end of a month; and, in cases still more chronic, a twelvemonth has been known to elapse before the patient finally recovered or died. Such are the general phenomena of acute and chronic glanders, as they have been observed in the human subject.

**Cause.**—The remote cause of glanders in the horse is but little understood. It is probably due to a specific miasmatic poison, having a peculiar affinity for the horse, and animals of his class. Glanders, however, when it affects the human subject, has in all instances been distinctly traced to the glandered horse as the remote cause. No instance is known of the disease occurring primary in man.

In the horse certain predisposing causes greatly favor, and are perhaps necessary to, the spread of glanders, such as dirty, close, ill-ventilated stables, especially if the situation be low and damp. Horses when crowded on board transports are greatly liable to this affection. The Arab, in transporting his horses from Arabia to India, always chooses that part of the year when the passage is shortest, lest the accidents incident to a long voyage might oblige the hatches to be closed, and want of ventilation promote the development of glanders. Bad food is a powerful predisposing cause in the horse, especially when these animals are picketed on service, and thus exposed to the inclemency of the weather. At the close of a campaign the cavalry are often decimated by this disease, and towards the termination of the Peninsular war the losses from this cause are said to have been enormous. The cases occurring in the human subject are too few to allow of any inference being drawn as to the influence of the predisposing causes in the production of glanders; but the disease generally occurs in young men; and probably a close investigation would have shown that the habits of the patient were such as to fall within those laws which favor the production of the disease in the horse.

The majority of veterinary surgeons, of stable-keepers, and coach proprietors, believe that the disease is contagious among horses, and if a glandered horse has been introduced into stables, the stock in these stables have become diseased. There are few districts in which some farmer, by the loss of a considerable part of his team, has not had sufficient proof of the communicable nature of glanders. In this country the law is severe against offering for sale, or even working a glandered horse; which shows that the opinion of our ancestors, time out of mind, has been that glanders is a contagious and a fatal



inoculation, I afterwards produced both glanders and farcy. In acute glanders, therefore, the blood is undoubtedly affected."

**Period of Latency.**—The poison of glanders has its period of latency, like all other morbid poisons, and that period is in general short. Two asses were inoculated by Mr. Turner, the one about a year and the other a year and a half old, and in the first the maxillary glands became tender on the second day, and the discharge from the nostrils was established on the third. In the other the maxillary gland enlarged on the third day, but the discharge from the nostrils did not take place till the sixth day. Sometimes, however, the incubation is much longer. In the *Procès-verbal de l'École de Lyon* a case is given of a horse which was inoculated with farcy matter, but the disease did not appear till the end of three months, and then precisely at the points of puncture. M. Gerard, an ex-veterinary surgeon of the French "artillerie de la garde," states that he introduced the matter of the discharge every day into the nostrils of certain horses, by means of a brush, and that the disease appeared in one on the seventh day, but in two others not till the thirty-second day.

In the human subject the poison has in general been latent from two to eight days after the accident of inoculation.

**Prognosis.**—Of fifteen cases of acute glanders collected by Rayer only one recovered. Of fifteen cases of acute farcy only five recovered. Of seven cases of chronic farcy only one died. Of the three cases of chronic glanders two died. A favorable prognosis consequently, is only warranted in the chronic form of the disease.

**Diagnosis.**—"Acute glanders," says Rayer, "cannot be confounded with poisoning from puncture in dissecting or opening dead bodies; for," he adds, "out of fifty such cases reported by various authors, no mention is made in them of a discharge from the nostrils, or of a nasal or laryngeal eruption being found after death, or of the peculiar cutaneous eruption." Leblanc also states that he has inoculated the horse with a great number of other morbid secretions from the human subject, but has in no instance produced any disease similar to glanders. It may for a short time be mistaken for rheumatism, but the occurrence of the secondary actions quickly dispels this error.

**Treatment.**—All the remedies hitherto tried in acute glanders have failed. The coming on of typhoid symptoms has led to the administration of *quinia*, *valerian*, *serpentaria*, *ammonia*, and other stimulating medicines; but all of them have failed. Vomiting and purging have likewise been had recourse to; but these measures have been equally unsuccessful. It is probable, therefore, that the cure of this disease depends on the discovery of a specific remedy, and experiments in treatment may be warranted as the only chance of subduing a malady which has so constantly proved fatal. In the more chronic forms of the disease the recovery of the patient has appeared to be owing to the excellence of his constitution during the natural elimination of the poison, to good ventilation, and to generous diet, rather than to any powerful effect produced either by general or local treatment.





poison seems to be even more virulent than it is in man. Death is more speedy; there is a more rapid spread of gangrene; and while the animal is yet living, the extrication of fetid gases from the tissues of the parts affected goes on to a great extent. The contagious property of the poison is possessed in the highest degree by the lymph contained in the characteristic vesicles, and, next to this, by that peculiar exudation which occurs in the areolar tissue of the affected part, and in that of various parenchymatous organs, and sometimes in the serous cavities of the chest and abdomen. The identity of the malignant pustule of man with the "*charbon*" of cattle has been satisfactorily proved by the fact that the disease when contracted by man, has been communicated back to the animal by inoculation from man.

[Inoculation of the human being with matter from a braxy sheep will produce malignant pustule, and the inoculation of the matter of malignant pustule of man in a sheep will produce braxy (RENAULT).]

It is only at the onset that the disease is a local one; but very soon general poisoning ensues, which is due to the after-diffusion of the morbid changes and products engendered in the part first affected. This is a very important point in the pathology of the disease, and with a view to successful treatment; for the early destruction of the diseased part by caustic not only prevents the development of the constitutional disorder, but in many cases issues in a perfect and speedy cure.

**Propagation.**—The disease may be communicated to man in the following ways: (1.) By direct inoculation, as in the case of butchers, farmers, skimmers, herdsmen, drovers, and others, in whom accidental inoculation with it appears to be an event of no uncommon occurrence in countries where "*charbon*" is most rife. (2.) By means of the skin, or simply by the hair of diseased beasts. Trousseau, for example, relates that in two factories for working up horse hair, imported from Buenos Ayres, and in which only six or eight hands were employed, twenty persons died in the course of ten years from malignant pustule. There are many other cases related by Dr. Budd, and some which clearly show that the virus of *malignant pustule*, like other contagious poisons, when once in the dried state, may retain its powers for an indefinite period of time. The disease may thus be propagated through contact with bones, hoofs, horns, and the fat and tallow of animals dead of the "*charbon*." (3.) The disease may be communicated by eating the flesh of animals killed while affected with it, as also by using the milk and butter of affected cows.

[There is a good deal of diversity of opinion and discrepancy of evidence with regard to the risk of eating the flesh of animals affected with malignant pustule. Ramazzini, Lancisi, Caillot, Enaux, Chaussier, Foderé, Gamgee, Rendle, and others, give many instances where the disease has been developed in such as ate of the meat of cattle suffering from carbunculous fever. Dr. Livingstone (*Travels in Southern Africa*), says, that those who eat the flesh of animals who die from pleuro-pneumonia,



which first appeared. These are at first isolated, but speedily they become confluent. The central spot may contain at first a transparent, bright yellowish fluid, which very early becomes reddish or bluish; then of a brownish hue, when the spot becomes extremely hard, very insensible, and rapidly becomes gangrenous. The inflammation extends to a considerable distance, both in depth and circumference; the neighboring skin is red and shining; the subcutaneous areolar tissue is puffy and emphysematous-like; the excoriated surface readily dries up, and becomes, as it were, mummified; and in its neighborhood new vesicles spring up, which run the same course as the former. The part soon loses its vitality, so that it may be pierced with needles without the patient becoming aware of it. It is also a remarkable feature of *malignant pustule*, that severe pain is generally absent. If the disease ceases to advance, an inflamed circle of vivid redness now surrounds the gangrenous portion, the tumefaction diminishes, and the patient experiences something like an agreeable warmth, accompanied by a pulsatory motion of the affected part. The pulse, which before was irritable and feeble, begins to revive, strength increases, a gentle perspiration indicates the crisis of the febrile state, and nausea ceases. Separation commences between the living and the dead parts, and is attended by copious suppuration. If the disease should not tend to a favorable issue, suppuration does not take place; the gangrene spreads rapidly; the pulse becomes smaller and more contracted; the patient suffers from extreme lassitude and inability to sleep; and, finally, with a tendency to syncope, he becomes passive as to the result. The tongue is dry and brown, the features shrink, the skin is parched, the eyes are glassy; and cardialgia and low delirium indicate the approach of the fatal termination (BUDD, RAJER, VIRCHOW, BELL, CRAIGIE).

The face (often in the lip, or immediate neighborhood of the mouth), the neck, the hands, the arms, and the legs, are almost the only parts on which it appears; and if by chance it becomes developed on other parts, we may be sure the poison has been carried there directly by the fingers, or other agents impregnated with the virus. The phenomena, therefore, which such cases exhibit in man are identical in every particular with those which have been seen in farriers, and others in continental countries who have the charge of cattle, and who in numberless instances are known to have become diseased from the accidental but direct inoculation of the "*charbon*" virus.

[*Malignant œdema of the eyelids* would seem to be identical in its nature and origin with malignant pustule (DEBROU,\* BOURGEOIS,† RAIMBERT‡). It begins with itching, quickly followed by great swelling, so that very soon after the onset the lids cannot be forcibly separated, owing to the degree of serous infiltration. The skin is tense and smooth, and

---

\* [L'Œdème Malin ou Charbonneux des Paupières. Par le Dr. Debrou. Gazette des Hôpitaux, No. 183, 1860. Arch. Gén. de Méd., Oct., 1865.]

† Traité de la Pustule Maligne. Par Bourgeois (d'Etampes).

‡ Traité des Maladies Charbonneuses. Par L. A. Raimbert. Paris, 1859.]



pears to preponderate in favor of *potassa fusa*; although Chaussier and others prefer *nitric acid* or the *chloride of antimony*. But everything hangs on the recognition of the disease in its first stage (BUDD).

[M. Manoury excises the pustule, and covers the wound with corrosive sublimate in powder. MM. Mauvezins cut out the "parent nucleus," or the entire vesicle, and then use the actual cautery at a red heat, and claim that, when resorted to at once, it hinders general toxic symptoms, and invariably cures (*Arch. Gén. de Méd.*, March, 1864, and June, 1866). Bourgeois relies on caustic potash, which he applies, removing the eschars, until blood appears. Most of the French surgeons depend on the actual cautery. It is probable that chromic acid, from its rapid and powerful action, would prove efficient, followed, after wiping away the detritus, by the free application of a strong solution of the chloride of zinc, 50 grs. to the ounce of water. As soon as the constitutional symptoms set in, alcoholic stimulants and carbonate of ammonia should be largely given, together with concentrated nourishment.]

## SYPHILIS.

LATIN EQ., *Syphilis*; FRENCH EQ., *Syphilis*; GERMAN EQ., *Syphilis*; ITALIAN EQ., *Sifilide*.

**Definition.**—*The result of a specific poison implanted on some part of the body, but generally through an abrasion or sore consequent on sexual intercourse with an infected person. A peculiar series of phenomena supervene, which mark the general infection of the system. The principal anatomical signs of general infection consist of induration (specific) round the spot where the virus has been implanted, induration of the lymphatic system of glands, the formation of nodes or gummatous nodular tumors in the connective tissue generally, and especially in that of the true skin, bones, mucous membranes, and solid visceral organs—e. g., liver, brain, lungs, and heart. A cachectic condition of the system accompanies the phenomena of infection; and indurations may remain in the form of hardened fibrous tissue in various parts of the body for an indefinite period of time.*

**Pathology and Morbid Anatomy.**—Advances in Pathology of late years have not been more marked in any direction than in demonstrating the very remote effects which syphilitic infection exercises upon the organs and the constitution of man. These advances are due to clinical, experimental, and post-mortem, observations. They have shown that a considerable number of doubtful cases of ill-health are in reality due to the specific poison of that venereal disease to which the name of *syphilis* is now restricted, whose morbid effects are not fully developed till many days, months, and even years after inoculation. Hitherto surgeons have claimed the subject of *syphilis* as their peculiar field; but after the surgeon had healed the sore, the morbid influence of the poison in many cases still remained, and internal lesions, impaired health, and degenerate constitution, eventually brought the patient to consult the physician as well as the surgeon.

The pure surgeon and the pure physician must, therefore, conde-



lief that "the venereal disease induces blindness, amaurosis, deafness, phthisis, rheumatism, epilepsy, mania."

There is no disease which more imperatively demands the careful study of the profession at this time, and especially of the army medical officer. The specific distinctions between the "infecting" and the "non-infecting" poison, and the characteristic phenomena they induce, are now being recognized at most of the continental schools. They are distinctions which are of great value in practice, and likely to become more valuable as our knowledge becomes more defined. Even now, indeed, when we see a *primary sore*, AND WATCH IT, we are able to predict with absolute certainty, at an early period of its development, whether the patient will or will not be the subject of secondary symptoms.

A history of syphilis in soldiers is too often the starting-point of a fatal disease. The impairment of the health takes its origin from the date of the *infecting* syphilitic sore. Early implication of the lymphatic glands leads to impoverishment of the blood as an immediate result, and then to the degeneration or wasting of tissues, which attends the general cachexia, and which eventually terminates in death, with complicated and varied lesions especially implicating the internal viscera.

No statistical Nosology gives any idea of the number of men lost to the public service from *syphilis*. The loss of strength from venereal diseases alone is equal to the loss of more than eight days annually of every soldier in the service. Dr. Balfour relates, in his most excellent and interesting *Medical, Sanitary, and Statistical Report of the Army Medical Department* for 1860, that "more than one-third of all the admissions into hospital have been on account of venereal diseases (369 per 1000); and the average number constantly in hospital is equal to 23.69 (reduced to 19.10 in 1864) per 1000 of strength (2315 men), each remaining in hospital on an average  $23\frac{1}{2}$  days. Thus the inefficiency is constantly equal to about  $2\frac{1}{2}$  regiments." Dr. Balfour also observed the individual history of 1126 men of the Grenadier Guards for three years and five months: 536 of these men gave rise to 1250 admissions; 212 were admitted *once*; 146 *twice*; 70 *three times*; 55 *four times*; 24 *five times*; 19 *six times*; 6 *seven times*; 2 *eight times*; 1 *ten times*; and 1 *fourteen times*.

[In 1861 venereal diseases caused a loss to the state, says Dr. Parkes,\* of a period equal to 8.69 days for every man serving at home; of nearly 89,000 men, there was a daily inefficiency from venereal of 2077 men. In 1862, the troops being 78,173 in number, there was a daily inefficiency with venereal of 1739 men. In 1862, of 8.12 days, or equal to the loss of two regiments constantly. In 1863, of 7.4 days. Dr. Balfour calculates that 60 per cent. of the reported venereal cases is syphilitic (recent or remote). In 1862 there were 7771 cases returned in the British army as "*syphilis primaria*," out of a total of 25,789 admissions from "*enthetic diseases*," or at the rate of 30.13 per cent. of the admissions from "*en-*

---

\* [A Manual of Practical Hygiene, by Edmund A. Parkes, M.D., 2d ed., 1866, p. 518.]





has been syphilis, as shown by the frequent occurrence of the marks of continued and dominant syphilitic action in their bodies; so that the influence of this cause is very imperfectly indicated by the number of admissions and service lost under the head of enthetic diseases (PARKES, MACLEAN, AITKIN).]

The Director-General of the Army Medical Department very properly requests that "all venereal ulcers be fully described, and the case fully kept;" and if such a request were complied with in the fullest sense of the term, most valuable results would accrue to science. From the nature of the facts and data about to be considered, the great importance of this request will at once appear; and the necessity of describing most fully, distinctly and clearly the origin, development and results of venereal sores, as far as possible, will appear obvious.

With a view to this accurate investigation and recording of results, the following points are worthy of notice:

1. The nature of the contagious principle of the syphilitic poison, as expressed in the opinions of the most trustworthy observers in this and other countries.

2. The characters and the phenomena which distinguish a sore that will contaminate or infect the system, and one which will not.

3. The vehicles or media by which the specific or "*infecting*" virus may be inoculated.

4. The secondary lesions and local growths in the internal viscera which are now so uniformly found to be associated with a history of syphilis, and which are the remote effects of a specific venereal poison.

1. **Nature of the Syphilitic Poison.**—The disease develops itself after the introduction of a specific virus; and the source of the poison is more distinctly traceable than that of the miasmatic order of diseases. The actual substance or matter which contains the virus can be obtained, and can be inoculated. Yet the *active principle* of the poison has not been isolated by any chemical process; and in this respect it is in exactly the same position as the poison of small-pox. The poison of syphilis undergoes a multiple process of elaboration or development in the system before its full effects are completed; and the lesions it induces demonstrate some of the most interesting points in the pathology of the multiplication or reproduction of morbid poisons. It is this multiplication which ultimately destroys life, through a general degeneration of the tissues and the establishment of a cachexia already referred to; or by the induction of grave lesions in important visceral parts, such as the brain, the lungs, the liver, or the kidney.

The earliest effects of the syphilitic poison upon the system become established during the occurrence of a "*hardening process*" which ultimately surrounds an infecting venereal sore—the local papule and its subsequent ulcer or sore. This hardening process is peculiar; and although not constant as to the local sore, it is constant as regards the glands or lymphatics which proceed from the vicinity of the part inoculated. It occurs in one or other of the



## 2. Characters of Venereal Sores, and especially of the "Infecting Sore."

—There are several independent affections to which the common

applied only to such cases as are believed to be of a *specific infecting* kind. Non-syphilitic venereal lesions are to be named according to their local and physical characters, as '*superficial abrasion*,' '*ulcus*,' and the like.

"2d. The following five points are to be noted in entering the history of each venereal case in the case-book :

"I. Physical characters and exact site of the lesions. II. Period of incubation. III. Character of attendant inflammation. IV. Effects on neighboring glands. V. Prognosis.

"3d. Under I. 'Physical characters and exact site of lesion,' state whether,

"(A.) The lesion has the appearance of a papule: fissure: an abrasion: of a dry, or moist, open sore; whether, if a *sore* exists, it is superficial, not appearing to penetrate the whole thickness of the integument: or deeper, with a smooth surface, scanty, chiefly serous secretion, grayish in the centre; whether the texture around the lesions is indurated, and, if so, what is the character of this induration, especially whether it is circumscribed, cartilaginous-like, and appears to be distinct from the subjacent and surrounding tissues; or whether,

"(B.) An excavated sore exists, with abrupt defined edges, involving the whole thickness of the integument, with an uneven surface, covered all over with copious secretion, and without circumscribed induration.

"*Mem.* : The induration which exists from simple inflammation excited by the rubbing of clothes, the probability of which the site of the sore will perhaps indicate, or by the use of irritating applications, such as nitrate of silver, &c., and which disappears gradually in the surrounding tissues, must be carefully distinguished from the circumscribed hardness characteristic of the true syphilitic sore.

"If more than one sore exists, it must be noted whether the several sores appeared together from the first, or appeared in succession.

"If some time has elapsed since the patient was first taken under treatment, the *original* form and appearance of the sore should be traced as far as possible, and noted whether it began as a pimple, abrasion, fissure, or otherwise.

"4th Under II. 'Period of Incubation,' should be ascertained and stated whether,

"(A.) The lesion first appeared after a lapse of one week, or from that time to a month, after exposure to contagion; or whether,

"(B.) There was no period of incubation, the sore appearing within a week after exposure.

"*Mem.* : The importance to the patient of the questions at issue should be frankly explained to him, and his confidence secured, so that he may be induced to state as exactly as he can the number of times he has been exposed to contagion within a period of four or five weeks prior to his discovering the existence of the lesion. A patient usually himself dates the origin of the lesion from the time when he was *last* in the way of contracting disease. He may, however, have been in the way of contracting disease many times *after* the particular occasion on which he really contracted it.

"5th. Under III. 'Character of attendant inflammation,' state whether,

"(A.) The inflammation appears to be of the adhesive, or whether,

"(B.) Of the suppurative or phagedenic kind.

"6th. Under IV. 'Effects on neighboring glands,' state whether,

"(A.) The superficial inguinal glands are, on one or both sides, generally and separately indurated, the inflammation with which they are affected being of an indolent character and without pain; or whether,

"(B.) The glands are free from enlargement; or whether one or more of the glands are enlarged, and exhibit a tendency to suppurative action.

"7th. Under V. 'Prognosis,' state whether you consider the case to be one of (a.) syphilis, or (b.) of local venereal sore, or (c.) of a doubtful nature.

"If the circumstances described under (A.) exist, the conclusion will be that the lesions are indicative of the constitution being affected by syphilis; if those described under (B.) exist, the conclusions will be that the lesions are local.

"If your prognosis is doubtful, state the considerations which cause it to be so.

"If the prognosis that the patient is afflicted with syphilis be correct, then the specific sore will not be capable of repetition on the same person by inoculation; if



discharge. The inoculation on the GLANS was followed by itching *fourteen days after the puncture was made*: three days later a speck appeared where the puncture had been made. The speck became a papule, then a pimple, and ultimately discharged yellow matter. The sore on the prepuce broke out several times after it healed up; but the sore on the glans never broke out again after it healed. The secondary lesions of syphilis followed this experiment, demonstrating the "infecting" nature of a virus with which he had been inoculated. Ulceration of the throat commenced in due time, and copper-colored blotches on the skin followed in the usual sequence. The time the experiment took, from the first infection to the complete cure and elimination of the poison, was three years.

Now, with the knowledge of syphilis which we possess, can we say from which of these sores the constitutional disease arose? The answer will evolve itself in the sequel. Hunter believed he had inoculated the discharge of a specific gonorrhœa *only and alone*; but two important questions now suggest themselves, concerning which Hunter does not enlighten us, namely: Had the person a concealed infecting chancre from whom Hunter took the virus? Was the patient suffering from constitutional syphilis at the time he had a gonorrhœa?

Besides Hunter, Carmichael in this country taught the same doctrine of a *single virus*;\* and Cazenave in France.

II. *The Period and Doctrine of Ricord*.—Ricord established, by numerous experiments repeated in various ways,—(1.) That the inoculation of gonorrhœal discharge by the skin is followed by no specific result; (2.) That *at least two*, if not *three*, distinct poisons exist—namely, *one virus* which would produce a gonorrhœa—another *virus* which would give rise to a specific ulceration, called a chancre. The ulceration of a chancre he observed to follow a very definite course. It commenced, as a rule, within twenty-four hours after the inoculation of the poison. A pustule formed, which breaking, a *soft* or suppurating chancre was the result. Ricord, however, eventually recognized *two* classes of chancres—the *soft* and the *hard*; but he described them as originating in the same way,—by contagion from a similar primary sore. His experiments were of one or other of two kinds. Either they were made on persons who had been already affected by syphilis—now known to involve a most

---

\* [Carmichael maintained the doctrine of a plurality of infecting viruses (*An Essay on the Venereal Diseases which have been confounded with Syphilis*. By Richard Carmichael. Dublin, 1814). He is claimed, though wrongly, by the dualists, as the father of their doctrine; for, besides several continental writers, including Swediaur, Abernethy (*Surgical Observations on Diseases resembling Syphilis*, 1804) speaks of contagious ulcers of the genital organs not followed by constitutional symptoms. The absence of induration of the base, and a tendency to slough, he particularly mentions as their chief characters. In the third edition of the same work (1814) he remarks, "I have never seen the phagedenic ulcer, which suddenly sloughs, affect the constitution." Carmichael divided ulcers on the genital organs into *syphilitic* and *venereal*, and of the latter he makes four subdivisions. (1.) Superficial, edges elevated, non-indurated; (2.) The same, without elevated edges; (3.) Phagedenic; (4.) Gangrenous. He expressly states, too, that these are sometimes followed by constitutional symptoms, and gives examples.—EDITOR.]



In women, compared with men, the open sore is said to be still more rare as the form of "infecting" sore. A hard chancre or sore in them is exceptional; and when it does occur, it remains small, is ill-developed, and is readily overlooked, even when searched for with great care, aided by a vaginal examination with the speculum.

[The induration disappears too at an earlier stage in women than in men, hence the *age* of the sore at the time of examination should be noted. The rapidity with which well-marked induration often passes away in the female, has been noticed by Clerc; he has also remarked that in certain regions it is more developed than in the same parts in men—as the lips and orifice of the urethra.]

In them the primary lesion which *infects* the system is always a papule (SIGMUND, CLERC). Another peculiarity connected with the "infecting" sore in women is, that such papules are apt to form along the course of the superficial lymphatics; and Ricord admits that induration is generally absent or ill-developed in primary sores in the vagina.

When the papule opens and becomes a sore, the fluid discharged from its open surface has been shown by Hubbenet, Lee, and Rollet to furnish a diagnostic test of the kind of disease, and of the sore from which it proceeds. Sigmund does not go so far as this. He does not consider the sores or chancres so different in form or character as to be at once distinguishable the one from the other. *He waits to see the virus produce PART of its effect* upon the system beyond the site of inoculation before he decides as to the nature of the sore. He waits to see the lymphatics indurate. He believes that then, and not till then, the distinction can be absolutely drawn between a sore which will infect the system and one which will not. He believes—(1.) That if induration of the lymphatics does *not* take place within six or eight weeks, and (2.) That if repeated successful auto-inoculation can be made on the bearer of the chancre during this period, then it is certain that the sore will not infect the system. If, on the contrary, the lymphatics indurate, and auto-inoculations cannot be then effected, the sore is assuredly an "infecting" chancre.

The addition to our means of diagnosis from the nature of the discharge—pus from the one, not from the other—is one of great value when it can be made, because the diagnosis as to the probability of subsequent infection may in some cases be made earlier.

**Period of Incubation.**—The time of the commencement of a sore or lesion after inoculation or contagion is of great importance to be noticed. A definite period of incubation exists for the "infecting" sore, fixed by experiment as well as by casual observation. Diday and Rollet fix the period at twenty-four days if the poison is from a primary sore; but at twenty-six days if the poison is from a secondary lesion. Sigmund, of Vienna, fixes the period of incubation at from fourteen to twenty-one days. Sometimes it may be longer, but never beyond six weeks or forty-two days. The circumstances which may protract the period thus long are exhausting fevers, pregnancy, and anæmic states of the constitution.





And let the unscarred braggarts of the war  
Derive some pain from you.

TIMON OF ATHENS, *Act iv, Scene iii.*]

The following table exhibits a scheme of the periods of appearance of the phenomena after inoculation from an infecting sore, and estimated from the first appearance of the papule or sore (SIGMUND):

			Day—9th	10th	14th	17th	19th	21st
I. INDURATION OF SORE, .			Cases—71	84	76	15	12	3
	Week—4th	5th	6th	7th	8th	9th	10th	11th
II. ENLARGEMENT OF GLANDS, .	Cases—31	44	56	74	46	20	13	9
III. SPOTS ON THE SKIN, .	"	—	2	41	68	45	22	11
IV. PAPULÆ AND PUSTULES, .	"	—	—	3	10	11	24	27
V. AFFECTIONS OF FAUCES, .	"	—	—	7	22	34	41	41

**Cutaneous Affections.**—Besides the general involvement of the glands, the condition of the skin may further demonstrate the contamination of the system. In the more insidious form of contamination its color generally is altered. It becomes pale, white, fawn, yellow, or brown; and is wrinkled, dry, harsh, rough, and hard, and no longer soft and elastic. The eruptions are papular, pustular, or scaly, and they are peculiar in their symmetry of distribution, and in the curvilinear character of their grouping. They leave behind them stains of color, pale cicatrices, or persistent ulcerations of the true skin. The local distribution of the syphilitic eruptions are also peculiar (DEVERGIE). "Their seats of election in the order of frequency are,—(1.) The parts round the alæ of the nose and the angles of the mouth; (2.) The roots of the hair at the forehead and back of the neck; (3.) The inner angle of the eyes; (4.) The centre of the breast; (5.) The inner side of the limbs, the neighborhood of the axilla and the groins."

**The Affections of the Fauces** are often not more than a peculiar color of the mucous membrane, persistent, however, like the staining of the skin (GAIRDNER), and eventually leading to disorganization. In women the process may cease with a slight follicular swelling of the mucous membrane of the fauces, tonsils, and soft palate (SIGMUND). If the process does not cease, then superficial erosions or deep ulcers of the soft palate supervene. Or still more diffused forms of ulceration may set in, involving great destruction of parts, and spreading in all directions—encroaching on the nasal fossæ and pharynx, eating away the epiglottis, extending down the air-passages, and even causing necrosis and exfoliation of the cartilages of the larynx.

**Second Attacks of Syphilis.**—The general infection is of such a kind as to render the system, as a rule, proof against a second invasion of the specific "*infecting*" virus. The disease never repeats itself, except, it may be, after a long interval. Sigmund has seen such a case. In this respect it resembles other virulent diseases acknowledging a specific virus as their origin; and in them the immunity is usually, but not invariably, complete—*e. g.*, small-pox, cow-pox, scarlet fever, and the like.

[This statement should not, in the present state of our knowledge, be



(2.) Mr. Lee has shown that there is a *certain condition* of the chancre in which, at any stage, on being inoculated or transplanted, it will produce a sore. It then appears to be autoinoculable. But this is only in appearance, and not in reality. The condition of the chancre that does this is one of *irritation*. Blister a chancre, or irritate it by an irritating ointment, or by any other means, so as to cause pus to flow—free pus-corpuscles being generated—and then we may have what has been recently termed a “mixed chancre,” of much more frequent occurrence than has generally been supposed (SIGMUND). Sigmund has produced such chancres by inoculation. The utmost caution, therefore, is necessary before pronouncing a sore to be *non-syphilitic*—i. e., “*non-infecting*.” Sigmund inoculated the pus of a soft, contagious, or suppurating sore upon the infiltration or sclerosis of a hard papule on which the skin had remained unbroken. Between twenty-four and forty-eight hours after, a suppurating ulcer was established, which afterwards assumed Hunterian characters. Inoculation of two poisons may thus be in some cases simultaneous or successive. Hence, “mixed chancres” present two aspects: on the surface is the soft, contagious, pus-producing ulcer; while deeper down is the specific syphilitic infiltration of the true “*infecting*” virus. Local plugging and enlargement of the superficial absorbents take place from such “mixed chancres,” followed by similar infiltration of the group of lymphatic glands nearest to the sore, spreading gradually to distant and more distant groups. This is the constant series of phenomena after syphilitic “infection,”—a regular series of connected events, giving rise to such symptoms as are associated with no other disease-poison except that due to syphilis.

In many cases of sloughing phagedena the syphilitic poison at once induces slough, as is the case with the poison of snake-bites, already referred to at page 647, when the tissue and the virus both die simultaneously. Such cases do not, as a rule, infect. So, also, if the part inoculated is made to slough by escharotics, both the virus and the tissue may be destroyed; and herein lies the value of the early use of caustic sufficiently powerful to destroy the inoculated part.

On the other hand, the sore which does not infect, and which does not contaminate, is the “soft,” suppurating sore—the “chanroid ulcer,” as it has been called—or “the simple contagious ulcer of the genitals.” The virus begins to act from the very moment of its application, and, after the formation of a pustule, ulceration is generally established by the sixth or eighth day from the time of infection, [or sooner.]

[The sore is characterized by active ulceration and suppuration, its edges and base are discontinuous, the vertical edge as it joins the base being a little undermined, and the former can be slightly moved on the latter, a little pus oozing out. The base is cellular and honeycombed, and the sore, when felt between the finger and thumb, is soft and doughy. There is never marked or well-defined specific induration. Rarely more than one or two inguinal glands are affected.]



[The received views on these points may be thus summarized:—(1.) A true chancre is an excoriation or ulceration produced by a specific virus at the point of deposition or inoculation, and is the antecedent of constitutional syphilis in the person so affected. (2.) There is a constant, though variable lapse of time between the inoculation and the appearance of the local sore—period of incubation—whose mean duration varies from twenty-two to thirty-five days. (3.) In a large majority of cases a true chancre begins with an erythematous inflammation at the contaminated point, which is immediately followed by a diphtheritic product *sui generis*, especially when its site is a mucous membrane. At times it is a flattened induration, or an enlarged papule, covered, perhaps, with an adherent scale (H. LEE). Induration of the base of the sore is very common, and is held by many to be pathognomonic; but this is an error, for it may not exist either as an early or subsequent symptom, as in chancre of the integument of the penis, and of the genital organs of the female; or it may be slight from the onset, and soon disappear; or it may not be detected; or specific induration may not be distinguished from hardness, the result of other morbid processes. Induration happens in most cases at the end of the first or beginning of the second week; it may appear later. It is fibro-plastic and presents different characters according to its age and the site of the sore. At the onset it is slight and diffuse; at the period of maturity of the chancre it is usually marked, deep, well-defined, hemispherical like a pea, and extends beyond the limits of the sore. In the ulcerous sore the edges slope inward to the base and are continuous with it. The typical form of true chancre may then be stated as beginning in a pimple or papule, which soon becomes an induration, and for some time is non-ulcerative, but covered with a gummy sero-epithelial secretion, and then taking on molecular disintegration at the surface. The induration is indolent, and is dissipated by a process of absorption, and not by liquefaction of the interstitial tissue-material. The formation of pus is an epiphenomenon from accidental irritation. A cicatrix is rarely left after the erosive form of true chancre. Four times out of five true chancre is single; if multiple it is so from the first. Except under circumstances of attendant irritation, the matter of a true chancre is not auto-inoculable, and then produces only pustulation and erosion without induration. Its duration varies accord-

---

means of a hand lens small circular elevations of cuticle are to be seen, raised by limpid serum. Several groups of these small vesicles generally occur, separated from each other by the space of a few lines. These eventually burst, and in their place small circular ulcerations, perfectly distinct, are to be seen, with a red bottom, and measuring scarcely a quarter of a line in diameter. The site of these herpetic ulcers is highly sensitive, and secretes pus and fluid, usually of an offensive and peculiar odor.

When the groups of vesicles are situated on the *cutaneous* surface of the prepuce, they are but slightly inflamed, compared with those situated on its internal or mucous surface. Frequently the fluid contained in the vesicles on the cutaneous surface is re-absorbed, when slight desquamation ensues over the lesion. If the fluid is not absorbed, it becomes opaque after a few days; and small scaly incrustations take the place of the group of vesicles. The disease may thus terminate in about *seven or eight* days.

When the groups of vesicles form on the internal aspect of the prepuce, they increase in size rapidly, and the inflammation is much more active. The walls of the vesicles are so extremely thin and transparent that the red color of the inflamed tissue may be seen through them. But the fluid soon becomes opaque and sero-purulent, small moist crusts or scabs form, which, being detached naturally or accidentally, expose excoriated spots, and it is important to distinguish these from soft venereal chancres. The venereal sores never commence as vesicles.



rightly, regarded as the first of the constitutional symptoms,\* “the prelude of the diathesis and the local reaction of the general poisoning” (WILKS). Not infrequently, after the local sore has lasted two or three weeks, rheumatoid pains, headache, weariness, &c., are complained of. These are early and sure tokens of systemic infection. They are very commonly followed, in the course of from four weeks to two months, by symmetrical exanthems on the skin and mucous membranes, symmetrical affections of the nails, hair, eye, and later, unsymmetrical ulcerations in the mouth, throat, and skin, tending to spread widely and deeply, with fibro-plastic exudations of the periosteum, connective tissue, muscles, fascia, nerves, viscera, not usually symmetrical, chronic in progress, and attended often with ulceration, or even a sloughing disposition, with tendency to relapse; for when the virus has entered the system, there is scarcely a tissue that may not be implicated, and that always in a specific and characteristic manner,—by the exudation of fibro-albuminoid material, modified to some extent by the organ in which it happens; in the solid organs as circumscribed masses, whilst on free surfaces it is seen on the base and borders of ulcerous sores, the same as in the primary local lesion. There is quite often entire freedom from any symptoms, lasting for months and even years, as if the virus had been exterminated; but usually certain reminders, in the form of scattered, scaly patches on the skin, as psoriasis palmaris, sores on the tongue, lips, &c., appear from time to time. So long as this tendency or state exists it is evidence of the presence of a virus in the system, communicable by direct or indirect means. Either from the prolonged effects of the special toxic agent upon the constitution, or from other concomitant causes, a cachectic condition may come on at a later period, varying from a few months to twenty years, with a tendency to fatty degenerations of the various structures of the body, and, perhaps, to those known as waxy or lardaceous. These are the so-called tertiary symptoms, but are more properly the sequelæ of syphilis. (6.) True chancre gives a *relative* and not *absolute* protection against subsequent attacks of the malady.]

**3. Vehicles or Media by which the Specific “Infecting” Virus may be Inoculated.**—Besides the discharge (non-purulent or mixed) from an infecting sore, there are at least three other sources of infection, namely,—(1.) The contagion of secondary syphilitic sores—*e. g.*, the syphilitic secondary ulceration of the female nipple, inoculating the mouth of the healthy infant born of healthy parents. (2.) It is now also established that *secondary* syphilitic inoculation (*e. g.*, the discharge from the softening and ulceration of gummatous tumors, mucous tubercles, papules, and the like) gives rise to a sore which exactly resembles a primary infecting chancre (LEE, ROLLET, VIENNOIS). But it is said to differ in the following particulars, namely,—(a.) The period of incubation is said to be somewhat longer; (b.) Ulceration is superficial; (c.) The sore heals in a shorter time; (d.) Induration is less marked; (e.) The constitutional infection is longer in developing itself; and (f.) The lesions which result are said to be not again contagious. Thus it is supposed the great epidemic of the fifteenth century gradually abated. Hence, also, perhaps, the modern belief in the modifying influence of syphilization may to some extent be explained—an operation which is not

---

\* [Ambrose Paré said, “If there is an ulcer on the penis and the part is hardened, it will be an infallible sign that the patient is affected with (constitutional) syphilis.”]





On the fiftieth day the color of the erythema became decidedly coppery, and treatment by mercury was then begun. In these experiments the blood communicated disease to *one* out of *five* who submitted to the experiment.

The recognition of this fact explains many occasional cases of syphilitic affection hitherto obscure—*e. g.*, syphilis from vaccination, contamination of a healthy nurse from the sore mouth of an infected infant, and the like. One of the most remarkable and lamentable instances of the inoculation of syphilis through vaccination is that which is now well known as the epidemic at Rivalta. At that place no fewer than *forty-six* children became affected with syphilis, the disease being communicated to each of them through the operation for vaccination (PACHIOLLI, SPERINO, *New Syden. Society Year Book*, 1861–62).

**4. Morbid Anatomy of the Secondary Lesions and Local Growths in the Internal Viscera.**—These are now so uniformly found associated with a history of syphilis that they are rightly regarded as the remote effects of the specific venereal or syphilitic virus. So varied are the effects of syphilis that a complete account of syphilis and its lesions has yet to be written; but it may be useful to illustrate some of the points of view from which the subject in its pathological bearings is now being examined, premising that it is necessary to *examine* the subject carefully from year to year, as opportunity offers and as fresh facts add to our knowledge, being ever alive to the fallacies which inevitably surround the most patient investigations.

[Syphilis in its ultimate form is capable of affecting every organ of the body; the internal organs may become equally as obnoxious to the effects of the virus as the external. Many obscure and intractable organic disorders are cases of visceral syphilis; and it cannot be too forcibly impressed upon the young practitioner, that syphilis may affect, “not only the cranium, but the brain within it, or the nerves; not only the muscles of the limbs and tongue, but the heart; not only the pharynx, but the œsophagus; not only the larynx, but the trachea, bronchi, and lungs; also the liver, spleen, and other viscera.” (S. WILKS, *Guy's Hospital Reports*, vol. ix, 1863).]

From any one of the sources of infection already noticed, the later stages of syphilis are characterized by lesions which are distinguished from the earlier specific affections, both by their situation and by their morbid anatomical peculiarities.

Differences in the stage of the disease have hitherto been mainly based upon the organs affected. The *primary affection* being local, the so-called *secondary affections* or *stages* were considered to be those which involved the skin, the mucous membranes, and the iris; while the *tertiary symptoms* or *stages* were those which implicated the areolar tissue, the bones, the muscles, the liver, the brain, the heart, lungs, and the kidneys.

A division based on the *anatomical* characters of the lesions seems to be more satisfactory than any arbitrary arrangement into stages of a supposed primary, secondary, or tertiary order (HALDANE).



form growths which lead to the development of elastic tumors, composed of a well-defined tissue, but with elements extremely minute. The gummatous tumor takes origin from the elements of connective tissue, or the analogues of such tissue, and hence the universality of the site of these lesions. They are like pus or an abscess in this respect.

When they first attract attention (as a node on the skin, or on the shin) they are small, solid, pale swellings, like a hard kernel, varying in size from that of a pea to the size of a haricot bean. They may be generally first seen in true skin, or subcutaneous or submucous tissue; and where the tissue is lax they grow to a considerable size, and give a sensation to the hand as if filled with gum. Repeated examinations of this growth show that in the gelatinous condition it arises from a proliferation of nuclei in the cells of the connective tissue—like the formation of granulations in a wound. The component cells appear as round, oval, or oat-shaped particles, embedded in a matrix of fine connective tissue, of a granular character, tending to fibrillation. The cells are a little larger than blood-globules, and contain granules in their interior when mature. In the young condition they are contained, and are seen to grow in groups, within the connective tissue corpuscles. In some respects they resemble tubercle, but differ thus in the mode of growth. How, then, do we recognize the nature of such growths? First, taking the history of the case as a guide, we are led to conclude entirely from the anatomical character of the growth; and when such lesions are seen in a case with a distinct history of syphilis, several questions suggest themselves for consideration. Is it the result of inflammation? Is it cancer or tubercle? Is it a syphilitic lesion? Are there traces of other similar lesions in the body? As a rule, *inflammation* leads to abscesses or hypertrophies of tissue or fibroid degeneration, and round all these syphilitic nodes we have such hypertrophy and degeneration, just as we have round tubercle nodules. Abscesses are easily recognized by the pus; and which, being altered by age, may still be anatomically recognized. *Cancerous* masses are recognized by the juice expressed from them. Here we have no juice; and the cell-elements seen in cancer are characterized by the diversity of their form and growth. Here the elements are uniform in appearance and size, and form growths less highly organized than cancers, which tend to infiltrate and involve neighboring textures; whereas the gummy syphilitic node remains isolated, and is usually surrounded by a dense but clear, semi-transparent, grayish, vascular, fibrous tissue, and very resistant to the finger. Thus these nodes appear sometimes as if inclosed in a kind of cyst, from which they may sometimes be enucleated.

By way of elimination or exclusion, therefore, we may thus come to recognize such growths as syphilitic—even without a history of syphilis (HALDANE). They have been recognized now and described in all the solid viscera of the body. The microscope has enabled us to study them with minuteness; but it is only their history, posi-



trices which result after absorption or elimination of gummata in bone have a characteristic appearance, especially denoted by the want of growth in the centre, and activity of growth at the edges after loss of substance.

2. The virus of syphilis seems to have the power of fostering the growth of simple interstitial inflammatory products, and thus lead to *hyperostosis*, *exostosis*, and *hard nodes*.

In the Skin there are two groups of syphilitic lesions to be recognized,—(1.) Local growths, which generally assume the form of eruptions; (2.) Cicatrices, [and, sometimes keloid growths.] The local growths occur in the superficial layer of the corion (VIRCHOW, BARENSPRUNG); and ultimately tend to grow deeper and to affect more permanently the derma and subcutaneous tissue (A. T. THOMSON).

When such growths soften (as they tend to do when superficial), great destruction of tissue is the result. The cicatrices which follow are permanent and unseemly, and may ensue without any abrasion of surface. This is especially the case in papular and tubercular forms of syphilitic skin diseases. The growth is generally associated with effusion of fluid, which causes the hardness; absorption taking place, atrophy of tissue follows; there is a falling in of textures, accompanied by obliteration of bloodvessels, and resulting in an unseemly white scar.

The diagnosis of syphilitic eruptions may in general be arrived at,—(1.) *From the history of the case.* A chancre commencing at least two weeks after exposure to contagion, becoming indurated, and followed within six or eight weeks by induration of the lymphatic glands. (2.) *From the symptoms accompanying the eruption.* A dusky tint of the skin, rheumatic pains in the head and joints, *alopecia*, ulceration of the throat, *iritis* nodes, *gummata*, disease of the testicle. (3.) *From the eruption appearing in several forms at one time on the body—e. g.,* Condylomatous, roseolous, lichenous. (4.) *From the general coppery tint of the eruption.* Strumous inflammations have a dusky red or vinous tint, simple inflammations in a healthy person have a bright red tint, but syphilitic eruptions in the chronic stage have a characteristic coppery color. (5.) As a rule, there is an absence of itching in syphilitic eruptions. (6.) *Syphilitic eruptions tend to assume a more or less circular form.* (7.) When the eruptions of syphilis ulcerate, the ulcers are generally round, with perpendicular edges and unhealthy bases. More than one of these characters must guide the diagnosis, and not one only.

[Nodes beginning in, and for awhile limited to, the connective tissue, are frequently met with in women, rarely in men. Their common site is the lower extremities, about the knee. They begin as a tender, hard lump, which as it grows becomes empty and soft, with a deceptive sense of fluctuation. The skin is soon adherent, and of a dusky red color. Ulceration takes place, and a large core, like soaked wash-leather (HUTCHINSON), is exposed, which is very indolent. These nodes happen from four to ten years, or longer, after the primary sore; and are not generally symmetrical.

The *bursæ mucosæ* are often implicated, when the tissues about them



of prolonged syphilis; and Virchow has frequently noticed lesions of the great vessels in those who die from syphilis with lesions in the brain. The tendency to aneurismal dilatations and cicatricial-like loss of substance in the lining membrane of the great vessels, in young subjects who are severely affected with syphilis, is a subject in morbid anatomy which requires yet to be investigated.

In the cases where cerebral symptoms have long coexisted with syphilis, "a quantity of tough, yellow, fibrous tissue unites together the surface of the brain with the adjacent membrane, and this again is adherent to the bone. The cortical substance of the brain at the affected spot is often partly destroyed, and the adventitious material occupies its place. The question has still to be solved as to what structure is primarily affected. Many have given the authority of their name to the opinion that the disease commences first in the bone, but simply for the reason that the osseous system is that which has so long been recognized as liable to be affected. But since we now know that other structures may be similarly attacked, we are prepared to look for its commencement in other parts, and even in the brain-structure itself. . . . . The cases which are so frequently met with are those where the deposit involves both sides of the *dura mater*, and includes in it the bone on one side and the brain on the other. The probabilities are in favor of its occurring in the *dura mater* first, as it arises in the periosteum on the exterior of the cranium" (Wilks in *Med. Times and Gazette*, Oct. 25, 1862).

But the lesion also occurs in isolated gummatous nodules in the great nervous centres, such as the *thalami optici* or *corpora striata*. I saw very recently (29th May, 1863) a most interesting dissection of such a case in the Middlesex Hospital, which had been under the care of Dr. Goodfellow. There had been a history of syphilis, and some of the children of the man had died of inherited secondary syphilitic lesions. A gummatous tumor occupied the left *optic thalamus*. Numerous cases of syphilitic tumor of the nerves and nervous centres are to be found collected together in the pages of the *Medical Times and Gazette*, and two may be referred to of the intra-cranial nerves, related in the 17th vol., for 1858, p. 419, in each of which paralysis was due to such syphilitic *neuromata*.

The lesions of encephalic syphilis are ushered in by obscure phenomena; but the following may be especially noticed: *Insomnia* manifests itself at the commencement; and *headache* is characterized by—(1.) Violence; (2.) Prolonged duration; (3.) Nocturnal recurrence or exacerbation. The *general nervous symptoms* are especially obvious in alteration of intelligence, of sensibility, and of motion. These, combined with such obvious local lesions as caries, or necrosis of the facial bones or of the cranium; or tumors on the external surface of the cranium, such as *gummata*, *periostitis*, or *exostosis*, at once point to cerebral syphilitic lesions; which are sometimes expressed by persistent epilepsy.

[The disorders of the nervous system caused by syphilis are innumera-





syphilitic hemiplegia is usually more favorable than that of other varieties; for the disease can be recognized betimes, and be judiciously treated.

Prof. Jaksch has seen twenty-five cases of syphilitic paraplegia, resembling very much locomotor ataxy. Also general paralysis, of which he makes three groups: 1. General paralysis, with mental symptoms like those of the disease commonly known by this name. 2. Loss of muscular energy, universally, without any affection of intellectual activity. 3. Palsy of all the extremities, with perfect power of the facial and ocular muscles, &c., and undisturbed mental activity. In the first form post-mortem examination shows especially either extensive softening of the cortical substance, or else atrophy of the same, with reticulated fibrous adhesions to the membranes. In some cases there was softening, with or without gummy deposits, partly in the anterior lobes, and partly in the central ganglia; in one case there was atrophy of the white substance of the hemisphere. In a case of the second variety there was softening of the pons, and thickly scattered deposits in the arachnoid of the brain and cord; in a case of the third variety there was softening of the cervical and lumbar portions of the cord, and no brain lesion. The age of the patients ranged from 23 to 58 years; only 5 were females. As a rule, the paralytic symptoms were only developed after several years' existence of constitutional syphilis. Only 5 cases were cured, and 1 improved.

It would seem that syphilitic paralysis may exist without any material lesion being revealed after death—*sine materiâ*—and that the poison may act directly on the nerve-tissue, producing molecular changes yet inscrutable, as well as by the development of intracranial or spinal growths in the osseous, fibrous, and connective tissues.\*]

Any form of syphilitic infection may be followed by nervous affections, from a year old up to old age. Syphilitic brain disease generally leads to *softening* of cerebral substance surrounding the nodule; and this softening cannot be distinguished from the softening induced by any other cause. The duration of syphilitic nervous affections averages about one year; and their natural course is characterized by intermissions; and at the outset the intermissions are very distinctly expressed.

[Ricord, Zambaco, and others, mention several cases of intermittent fever in which no progress in treatment was made until the syphilitic taint was suspected, and special remedies given.]

A point in the pathology of syphilis at present assuming considerable importance, is the influence which an open suppurating sore of a secondary or tertiary kind has in removing the tendency to the localizations of lesions elsewhere, and especially in internal parts. Dr. Steenberg says, with reference to cerebral lesions, that he has seen the existence of an ulcer of a tertiary kind act as a natural issue in subduing the irritation of cerebral lesions, an entire remission of the nervous symptoms occurring while the ulcer remained open. Hence the great benefit which often follows the use of a seton in syphilitic epilepsy.

---

\* [Des Affections Nerveuses Syphilitiques, par D. A. Zambaco, Paris, 1862.]



phthisis in those especially constitutionally predisposed, and where mercury has been taken. Periostitic thickening of the clavicle and the upper ribs is apt to lead to the belief that tubercle is present, on account of the consolidation. Care is necessary in the case of young soldiers, who, having been just discharged from hospital, after the cure of an infecting chancre, may be exposed to cold and wet on guard, and so have an attack of syphilitic bronchitis induced—the probable commencement of a growth of tubercles in the lungs, or of solitary syphilitic gummata (*Annual Report of Army Med. Depart. for 1861*). (2.) Deposit in the pulmonary substance, in the form of gummata, of the same histological constitution as the well-known subcutaneous product, which has been described by M. Ricord and McCarthy as forming in the lungs, especially towards their periphery and bases. Towards the periphery they are like nodules of lobular pneumonia. They soften, and are eliminated very much like tubercle, and have at first a consistence like scirrhus. They are non-vascular (WALSHE).

[**Syphilitic Lesions of the Œsophagus, Stomach, Intestines, Rectum, and Diaphragm.**—Syphilitic ulcerations in the pharynx may extend to the Œsophagus, and cause contraction at the upper part of this tube (WILKS). M. Cullerier, several years since, published his observations of a form of enteritis, accompanied with obstinate diarrhœa, in which submucous nodular deposits not going on to ulceration, were found. It was most common in children, though he had met with it in adults. There is no doubt that syphilitic patients are liable to dysenteric symptoms, which yield to special treatment. These are, however, generally due to ulcerations of the rectum, extending high up, as far as the finger can reach, not accompanied by condylomata, happening as a late constitutional symptom, and curable by suitable treatment (HUTCHINSON, PAGET, WILKS). Murchison and Moxon have each reported a case of syphilitic formation in the diaphragm.]

**Syphilitic Lesions of the Liver.**—Dittrich and Gubler were the first to give an accurate description of these syphilitic deposits in the liver. Virchow has also described a peri-hepatic lesion and a simple gummy interstitial hepatitis. The former never occurs alone, but is generally associated with the latter. The hepatic substance atrophies, and the deposit contracting, is eventually absorbed, causing a cicatrix-like mark. The liver lesions are usually among the later symptoms of syphilis, and are well described by Wedl, Virchow, Wilks, and Frerichs [Loudet and Moxon. See *Syphilitic Disease of the Liver*, vol. ii, by the Editor.]

[**Syphilitic Lesions of the Spleen and Pancreas.**—The *spleen* is liable to the syphilitic deposit, though to a less degree perhaps than the liver. It may occur as diffuse hyperplasia, or in pale nodular patches, with cicatriform indentation, which latter, according to Beer, are never found in other disorders. Moxon (*History of Visceral Syphilis, Guy's Hosp. Rep.*, 1868) has met with sulphur-yellow nodules, of the size of peas, plentifully scattered, deep-seated, and fatty in their centre. Gummata have been found in the *pancreas*.

**Syphilitic Lesions of the Kidneys.**—These may occur in two forms,—



**Hints for the Investigation and Description of Syphilitic Ulcers.**—1. Ascertain as near as possible the date of contagion, keeping in view the media or vehicles of contagion, in addition to virus from a true primary chancre—namely, from ulcers in acute secondary syphilis; from the blood of patients suffering from acute secondary syphilis; from sexual intercourse, followed by conception, with a man who is at the time suffering from syphilis in some of its active secondary forms; from mixed chancres carrying the virus; from sloughing sores carrying the virus.

2. Examine the patient, keeping in view,—

(a.) That the soft, “non-infecting” sore commences almost immediately (*i. e.*, twenty-four hours to within three days after connection). It commences as a red spot, or a point, passing very soon into a pustule and a soft suppurating sore.

(b.) That the “infecting” sore does not commence before the end of the second or beginning of the third week (eighteen to twenty-four days); and if the disease has been contracted from a secondary ulceration, not before the expiration of the third or fourth week (RINECKER). A specific sore results in the form of a papule, abrasion, fissure, or crack; the formation of pus, or an ulcer discharging pus, being an accidental occurrence.

3. Examine microscopically the discharge from all syphilitic sores, keeping in view—(1.) That a “soft, non-infecting” sore discharges pus-cells; (2.) That the fluid discharged from an “infecting” sore is not pus, but a molecular debris.

4. The irritation of an “infecting” sore may cause it to discharge pus along with the “infecting fluid.” Hence “mixed chancres.”

5. The soft, purulent, non-infecting sore may be transplanted at will, and at any time, on the patient’s body. The true “infecting” sore cannot be multiplied after glandular enlargement and general infection become developed. It remains a solitary sore.

6. Look every day for cutaneous eruptions during the existence of a primary sore.

7. Examine the lymphatic glands—not only in the vicinity of the chancre, but also those in the axilla, and the neck up to the occiput. Note as to the slowness or rapidity of the enlargement, hardness or softness, tendency to suppuration, and whether painful or not.

8. From the account of syphilis and venereal sores which has been given in the preceding pages, it must appear clear that definite nomenclature must be adhered to in describing venereal or syphilitic sores.

[The Venereal Commission already referred to, from the evidence before them, came to the conclusion, that the syphilitic sore is seen under three forms: one characterized by induration throughout its entire course; one soft in its early stage, and becoming subsequently hard; and one soft through its whole course, but which, unlike the simple local sore, is followed by constitutional disorder.

The evidence, they state, is conclusive as to the impossibility of pronouncing with certainty upon the character of a sore on its first appear-



persons suffering from syphilis by virus from a chancre, and repeated the inoculations once or twice a week till the virus ceased to produce any effect; and when this point was reached, all other sores had healed. This naturally gave rise to the belief that, like vaccination, the system became protected; and to this process the name of Syphilization has been given.

When we consider the suffering, the long confinement, the filthy sores, and the innumerable cicatrices left, as well as the doubtful results, the uncertain state of our knowledge regarding the virus of syphilis, and the media of its conveyance, it cannot be conceded that we are warranted in sanctioning the method of treatment by syphilization. At Copenhagen, at Florence, at Turin, and other places where large hospitals exist, extensive experiments have been carried out in public; and although time and additional evidence are both wanted to learn ulterior results, nevertheless I think the facts are capable of a totally different explanation from that which has been given them, and to which I have adverted. But let me go more into detail.

Sperino's cases and Lee's experience show that during the active existence of phagedenic suppurations and continuous suppurating sores, in a patient suffering from syphilis, the phenomena of secondary infection do not advance, but the symptoms of contamination gradually wear out. Moreover, suppurations are easily established on the syphilitic. The action set up in them by repeated inoculation—the so-called syphilization—is merely a continuous suppurative action: indurated sores are not produced. The system is already contaminated; and the infecting virus will not produce any additional specific effect. Lastly, syphilis, in course of time, tends to wear itself out of the constitution. Hence the *modus operandi* of so-called syphilization may be explained, conjointly—(1.) By lapse of time; (2.) By continuous suppurations affording a drain or source of depuration to the system; (3.) From simple non-specific ulceration being sufficient to accomplish this result, as shown by the fact that the experiments on syphilization have been effected from all forms of venereal sores, discharge having been taken indiscriminately from soft as well as true infecting chancres. Moreover, the experiments recently made by Dr. Lindwurm, of Munich, clearly prove that any curative influence which the so-called process of *syphilization* may possess is due to the excretory action of numerous and prolonged simple ulcerations. He submitted fourteen syphilitic patients to friction with tartar-emetic ointment, without any other treatment. When the pustules from one inunction had dried up, a fresh crop was produced by a second inunction in another place; and this was repeated. The results were, in some instances, surprisingly favorable, in others less good, and in others negative. He therefore justly considers that syphilization and tartar-emetic-ointment frictions produce like results (*New Syden. Society Year-Book*, 1860, p. 325).

The process to which the name of Syphilization has been given consists of the following details:

(1.) Matter is taken from a sore—an indurated one by preference.





yield pus, it was made to do so by slight artificial irritation. He inoculated thirty-six times with pus obtained from as many hard sores, and forty-three times with that from ulcerating mucous patches, and in every case with negative results. He states that Prof. Pellizzari, in 1865, also made a similar series of experiments, and with like effects. Dr. Gustavo Bargioni communicated to Dr. Ricordi the results of Bidentkap's experience in Paris. He says, he made between seventy and eighty experiments with the secretions of the indurated sore, but he never succeeded in producing a characteristic ulcer (*Annali Universal. di Med.*, Jan. 1866, quoted by H. Lee, *Lancet*, April, 1866). Mr. Lee says, that in all the experiments he has performed, where due precautions have been taken, he has always failed to produce a lineal series of inoculations from an uncomplicated indurated sore; and Dr. Boeck, he adds, did not, during his residence in England, succeed in producing such a result in any one instance. Mr. Walter Coulson has had two cases in which, after repeated inoculations, he obtained well-developed pustules. There was a possible source of error in one case, but in the second none could be detected.

The following conclusions of Mr. Henry Lee may be fairly adopted:

1. That no evidence has hitherto been adduced satisfactory to the profession that the infecting form of syphilis can be inoculated upon a patient who is at the same time the subject of constitutional syphilis.
2. That both from a soft sore, and also occasionally from the surface of a hard sore, matter may be taken, which may be made to produce a number of local specific ulcerations having the characters of the soft chancre.
3. That during the continued irritation of such ulcerations, the manifestations of secondary syphilis will disappear.
4. That the treatment of syphilis in this way is tedious, and inconvenient (*The Lancet*, April, 1866).]

**Treatment of Syphilis.**—As in the case of other *enthetic* diseases, it is clear that if the inoculation of the syphilitic virus could be recognized in time, the site of inoculation, and with it the virus, by being destroyed, subsequent infection of the system might be prevented. But experience shows we must not conclude that even by an early destruction of the sore the occurrence of constitutional infection will be always prevented. The exact nature of a sore cannot yet be recognized at a sufficiently early date (apart from all other means of diagnosis) as to whether it will or will not prove a sore carrying a virus which will infect the system. In cases where the sore is a suppurating one, *occurring late after exposure* to infection, such a sore may be of a mixed nature, and therefore is of doubtful character, and always suspicious.

The local progress of such sores may be arrested with escharotics, if they are applied at an early period of its existence, and before contamination of the system is evinced by induration of the base of the sore. Ricord and Sigmund have found that sores destroyed by the more powerful caustics, within *from three to five days*, have not been followed by syphilitic symptoms. But these may have been cases of soft chancre, which would not infect. The only efficient caustics for this purpose are—(1.) The *strong nitric acid*; or (2.)



destructive local affections. The non-mercurial treatment is slower, but surer; starvation and Zitmann's\* decoction being the means he employs. He believes that the proportion of cases of constitutional syphilis to those of chancre has greatly diminished since mercurial treatment has been discontinued (*Ann. de Berlin Charité*, ix, 1, 1860; *Syden. Society Year-Book*, 1861). Herman has come to similar conclusions, from his experience in the syphilitic wards of the Vienna Infirmary. He believes that the non-mercurial treatment is much more speedy and successful than the mercurial; that no relapses occur; and that cutaneous eruption is much more frequent and severe in patients who have taken mercury. The experience of Diday is not less decided. He states that mercury cannot now be said to cure syphilis radically, so as to render all relapse impossible. Its warmest advocates do not in the present day, claim more for it than the power of delaying only the appearance of the first syphilitic manifestations, and of hastening the disappearance of certain other lesions. He imputes to it positively, and on sufficient clinical evidence, the following disadvantages: 1st. It tends to render the primary ulcer phagedenic; 2d. It tends to induce stomatitis and necrosis of the alveolar borders; 3d. It produces an acute affection of the gastro-intestinal mucous membrane, and dyspepsia; 4th. It brings on trembling of the extremities, apoplexy, and insanity. All of these results he has seen supervene, even when the treatment by mercury was superintended and directed by the most competent and attentive practitioners.

He does not, however, withhold mercury in every case. If the primary lesion becomes an indurated *woody* chancre, mercury is to be given. If the chancre is a doubtful one, he recommends waiting till some of the early constitutional phenomena render the nature of the case evident, and indicate the probable gravity of the syphilis with which he has to deal. He employs *iodine*, *iron*, and *quinine*, on the appearance of slight relapses, with a tonic and supporting regimen. He recommends *iodides*, to combat the *chloro-anæmia*, and to relieve the pain of tertiary ulcerations.

Numerous examples may be seen in museums, which show that the poisonous effects of mercury produce the worst lesions of the two; and, when combined with the syphilitic virus in a strumous person, the worst of all. In the extreme of syphilitic infection, it ought never to be forgotten that a specific chlorosis results from syphilis, amounting to anæmia; and that mercury will bring about a similar anæmia; while numerous instances are quoted by authors,

---

\* Zitmann's decoction is of two kinds, the stronger and the weaker. The former is compounded as follows: R. Rad. Sarzaparillæ, ℥xij; Aquæ, ℔xxiv; Coque per horas duas et adde Aluminis, ℥iss.; Hydrarg. Chloridi mitis (calomel), ℥ss.; Antimonii Oxy-sulphureti, ℥j; *misce.* Coque ad  $\frac{3}{4}$ , et adde Fol. Sennæ, ℥iij; Rad. Glycyrrhizæ, ℥iss.; Sem. Anisi, ℥ss. *Infunde per horam et cola.* The dose of this decoction is half a pint to a pint morning and evening.

The weaker decoction is compounded as follows: *Cupiat residuum* decoction fortioris et adde Radicis Sarzaparillæ, ℥ij; Aquæ, ℔xxiv; Coque per horas duas et adde Cort. Canellæ, Cort. Limonum, Sem. Cardamomi, aa ℥iij. *Infunde per horam et cola.* The dose of this decoction is one pint at intervals during the day. [Mercury is the active ingredient in this preparation.]



given in the treatment of the local sore, its administration will not *prevent the occurrence* of constitutional symptoms; nevertheless, the value of mercury in the cure of THE INDURATION of the true *infecting chancre* is now fully recognized. The local lesion, if it appears after the usual prolonged period of incubation, is as much a manifestation that the constitution is already affected as is the developed vesicle of *variola vaccina* a manifestation that the constitution is affected with variolous poison. Looking, also, to the nature of the virus of syphilis, as expounded in the text (especially at page 677), the *excision* of the *primary lump* or sore—the *specific induration*—as practised by Dr. Veale (*Edin. Monthly Journal*, July, 1864), and by Dr. Humphry, of Cambridge (*British Medical Journal*, August 13, 1864), is a justifiable operation; for the original sore, when it has become a “lump” (as in its state of “woody-like” induration), is an undoubted maintainer of infection and of contamination of the system. If, therefore, it can be easily and completely insulated, as when on the *prepuce*, the cure of the constitutional symptoms may be facilitated.

There are also certain forms of secondary syphilis for which the administration of mercury is unsuitable. These are the pustular eruptions, or ecthymatous states in rupia and in syphilitic anæmia. For the cure of other secondary symptoms mercury is certainly of service. If given to the extent to which I have limited its use in the text, I believe that secondary symptoms disappear more rapidly under its regulated use than by any other plan of treatment. All our treatment of syphilis rests on that evidence which must always guide the hand of the physician, namely—practical experience. That has certainly taught us that the mercurialism of John Hunter’s time was an error; and that its regulated administration in cases of *syphilis* is undoubtedly beneficial, and especially during the evolution of the specific symptoms of infection. Dr. Jeffrey Marston, of the Royal Artillery, has given an admirable summary [*British Medical Journal* of Feb. 21, 1863] of the means and indications of treatment by mercury which he has found most useful. His experience shows that the system ought to be affected as slowly as possible; and there ought to be a remission of the remedy for a time as soon as that effect has been attained.

[The antidotal, curative, or specific property of mercury in syphilis may, perhaps, be not proved; but no medical man of large opportunity, free from partisanship, and who has fairly tried both the mercurial and non-mercurial practice in true syphilis, will deny that, when properly administered, mercury hastens the healing of the primary sore, abates the induration, lessens the liability to the happening of constitutional phenomena, removes these when they appear, and in many of the syphilitic sequelæ, when the dyscrasy is fairly established, with tissue contamination, it often produces marvellous results, after other remedies have failed. Hebra and Zeissl, after giving a fair trial to all the different plans of treatment—extractum graminis (expectant), subcutaneous injections of cold water, iodine and its preparations, syphilization—have come back to mercury (*Brit. and For. Med.-Chir. Rev.*, vol. xxxv, 1865). Mr. Hutchinson, though denying that it is an antidote, and believing that there



best treated by a combination of *liquor arsenicalis*, solution of *bichloride of mercury*, in very small doses, and tincture of *sesquichloride of iron*; while the use of soap in ablution ought to be avoided (STARTIN).

[R. Hydrarg. Chlor. Corrosiv., gr. j; Potass. Iodidi, gr. xxx; Liquor. Potass. Arsenitis, ℥xxxvj; Alcohol, fʒj; Ext. Sarsaparillæ, fʒiij; Aquæ Cinnam. ad fʒxij. Two tablespoonfuls three times a day after meals.]

In the administration of mercury for the cure of syphilis, *salivation*, or anything approaching to that condition, should never be induced. *Tenderness of the gums* should be the utmost physiological effect very gradually and gently brought about. The *primary sore* should be healed without mercury; but so soon as the evolution of constitutional symptoms has commenced, such as the specific induration of the sore or glands, cutaneous, scaly, tubercular, condylomatous affection, or iritis, the sooner mercurial treatment (to the extent indicated) is commenced the greater will be the benefit. But where *suppurative lesions* have been established, mercury ought to be withheld altogether, or very sparingly combined with *iodide of potassium*.

*Iodide of potassium*, in doses adjusted to the individual case, appears to act with rapid benefit in some of the syphilitic diseases of the interior of the cranium giving rise to extreme pain. Its administration often causes intense suffering in patients who have been treated by mercurials. Two distinct effects are produced: *first*, the compounds of mercury fixed in the body are rendered soluble and active; and, *secondly*, a form is given to them which allows of their elimination, with more or less rapidity, in a state of combination with one of the elements of the iodide; and thus the patient is subjected anew to a mercurial treatment by the compounds of mercury already present in his body (Melsens in *Brit. and For. Med.-Chir. Review*, 1853). The dose of *iodide of potassium* should at first be small—not more than *fifteen grains* in the twenty-four hours—increasing the dose, if the patient bears it well (MELSENS, GUILLOT). Its action is aided by a blister over some portion of the shaven scalp, and by having the blistered surface dressed with mercurial ointment; and, generally, it may be said that local treatment gives very valuable aid. For example, cutaneous or mucous *raised papules* remaining persistent, an ointment composed of *oxide of zinc*, *calomel*, and *simple cerate*, hastens their absorption. Eruptions of lichen, acne, and herpes are similarly benefited by the application of *oxide of zinc lotion ointment*; and if *prurigo* and *urticaria* be also present, *diacetate of lead lotion* will expedite the cure. Vesiculo-crustaceous spots will cease to reappear if the affected parts are painted for a few days with a solution of *nitrate of silver* (gr. x–xx to ʒj), [or chloride of zinc in solution, or carbolic acid, or corrosive sublimate], and *oxide of zinc lotion* applied afterwards.

In the more advanced stages of the suppurative affections, the use of pyogenic counter-irritants ought not to be neglected, such as tartar-emetic ointment. They tend to keep up just so much of a





where other modes of treatment, including the internal administration of mercury, have been used and failed. It may be given where the strength is much reduced, provided there is no organic visceral disease.]

The mercurial vapor bath is to be managed in either of the following ways. The first method is best adapted for the practice of a large institution; the method recommended by Mr. Lee is better suited for private practice:

“The patient is seated on a chair, and covered with an oil-cloth lined with flannel, which is supported by a proper framework. Under the chair are placed a copper bath, containing water, and a metallic plate, on which is placed from one to three drachms of the bisulphuret of mercury, or the same quantity of the gray oxide, or the binocide of this metal. From five to thirty grains of the iodide of mercury may be employed, or a scruple of the iodide, with a drachm and a half of the bisulphuret. Under the bath and plate spirit-lamps are lighted. The patient is thus exposed to the influence of three agents,—heated air, steam, and the vapor of mercury. At the end of five to ten minutes perspiration commences, which becomes excessive in ten or fifteen minutes longer. The lamps are now to be extinguished; and when the patient has become moderately cool, he is to be rubbed dry. He should then drink a cup of warm decoction of guaiacum or sarsaparilla, and repose for a short time” (LANGSTON PARKER).

Mr. Henry Lee's mode of proceeding is more simple: “A special and convenient apparatus is used [made by J. Ronchetti, 92 Fulton Street, New York], which consists of a kind of tin case, containing a spirit-lamp. In the centre, over the flame, is a small tin plate, upon which from fifteen to thirty grains of calomel is placed, while around this is a sort of saucer, filled with boiling water. The lamp having been lighted, the apparatus is placed under a common cane-bottom chair, upon which the patient sits. He is then enveloped, chair and all, in one or more double blankets, and so he remains well covered up, for about twenty minutes, when the water and mercury will be found to have disappeared.”

[There are several other points about the mode of administration of the calomel vapor bath, which are particularly insisted upon by Mr. Lee. The object of depositing the vapor of calomel on the skin is, first, to excite an action there, and secondly, that some of the mercurial may be absorbed. That this should take place, it is necessary that the powder should remain on the skin, and that the skin be soft and clean. If it is greasy, or covered with dry scales, its absorbing power is lessened, and the baths may be taken without any sensible effect. To insure proper action in the skin free perspiration should first be produced by the hot air bath—the first stage of the Turkish bath—and when the skin acts freely, the patient should be rubbed down, and immediately placed in the calomel bath. The action of the bath is materially assisted by the inhalation of a certain amount of the vapor.]

The corrosive sublimate (*Hydrargyrum corrosivum sublimatum*) is perhaps the next best form of administration; and where it has to be continued over many weeks, may be given in the following form, with opium (TANNER):



sur leur corps." Since then most syphiliographers have treated of hereditary syphilis, though Hunter always denied it, whilst he reported two cases which were undoubtedly of inherited origin. It is, however, only within a short period that it has been understandingly studied, and its several expressions at different periods of life satisfactorily made out, particularly the morbid changes which happen in the viscera. For the precision and extension of our knowledge on this subject, we are mainly indebted to Trousseau and Lasègue, Cullerier, Gubler, Nat. Guillot, Desruelles, Diday, Bärensprung, Förster, Henri Roger, Hutchinson, and others.

In all stages of Constitutional Syphilis the taint may be transmitted to the child. The degree of severity of the inherited taint is probably in proportion to the shortness of the period which has elapsed since the presence of active symptoms. A child may inherit syphilis in a serious form from but one parent—from its father alone, or from its mother alone. When both parents are the subject of syphilis, the child is more certain to suffer severely than when only one is so. There are as yet no sufficient data to form an opinion as to whether a child is more likely to be gravely affected when its father is the source of contamination, than when it derives the disease from its mother, or the reverse. In a large proportion of the cases met with in practice, the taint is derived from the father only (HUTCHINSON). Though infection of the offspring by the father is still contested by some authors, the numerous and positive cases collected by Bärensprung, E. Vidal, and others, put beyond doubt that constitutional syphilis of the father is transmissible to the child; but when he is suffering from the sequelæ of syphilis, the so-called tertiary stage, it rarely or never is. During the period of latency, often a protracted one, when there is no apparent manifestation of the disorder, he is capable of tainting his offspring. Bärensprung reports fourteen cases where this happened, and Diday cites several observations in support of this view. Mr. Hutchinson gives several facts in his paper published in the *London Hospital Reports*, vol. ii, p. 184, and remarks (Reynold's *System of Medicine*, vol. i, p. 299): "It is very common for a man who does not himself display a single symptom of any kind, and who appears to be in perfect health, to beget a syphilitic child, the symptoms displayed by the child being usually those of the secondary class. There is no doubt that the nearer the occurrence of the primary symptoms in the parent is to the birth of the offspring, the more certain is the latter to show symptoms of a severe character, and typically secondary in stage. Instances, however, are met in which infants, born ten years after the original disease in the parent, still display first a secondary rash, with the characteristic snuffles, &c. In several instances I have known a whole family of children, born during a period of from five to ten years, display each one the characteristic and transitory rash soon after birth." If the father alone is affected at the time of procreation, it is clear that the sperm must be the vehicle of transmission; but when the mother alone is diseased, the mode of infection of the child becomes a question. Is it through the blood, or by the ovum? Though it is contended by many that the sole agent is the blood, others believe the toxic matter to exist in the ovum, and to be subsequently developed along with it. Neither theory is supported by direct proof; analogy would favor the ovular theory, for, as Lancereaux remarks: "In view of the difficulty of the inoculation of the blood, and from the great likeness of the properties and the characters of the secretion of the testicles and that of the ovaries, there is more reason to believe in the influence of the ovum than of the blood. The hereditary transmission of



it is not the pallor, nor yet the icteric or strawy hue, of the other cachexiæ; it hardly extends to the rest of the body. The eyelashes are not developed, or have fallen out; the eyelids are often everted, and at the external angle are fissured. In the place of the missing eyebrows, there are yellowish scaly stains, which are sometimes found about the chin and mouth" (*Clin. Méd.*, &c., vol. iii, 1865). The child is generally fretful and cryful; sleeps but little; is troubled with vomiting and diarrhœa; and very liable to serous inflammations, as pleuritis and arachnitis, which are frequent causes of death. Erysipelas and pneumonia are common intercurrent disorders, and are generally fatal. The child sometimes dies in a state of extreme marasmus.

When the syphilitic symptoms are present to any extent at the time of birth, it is rare for the infant to live beyond a few months.

In those cases of hereditary syphilis which survive the first year, all traces of the disease disappear about that time, except perhaps unusual paleness, and an expanded nasal bridge, caused by long-continued swelling of the parts within. During the period of latency there is usually excellent health, though Mr. Hutchinson asserts condylomata sometimes reappear; but there is scarcely ever a return of the cutaneous rash. The third epoch may begin at any time after the fifth year, but it is commonly delayed until at or near the period of puberty. It is characterized by the lesions known as the tertiary stage in the acquired disorder. The diagnosis of inherited syphilis, at or after the age of puberty, may sometimes be made with much certainty, or it may be surrounded with great difficulties. "Our most valuable aids," says Mr. Hutchinson, "are the evidences of past disease, more especially of the inflammations which may have occurred in infancy. A sunken bridge of nose, caused by the long-continued swelling of the nasal mucous membrane when the bones were soft, a skin marked by little pits and linear scars, especially near the angles of the mouth, the relics of an ulcerating eruption, and a protuberant forehead, consequent upon infantile arachnitis, are amongst the points which go to make up what we recognize as an heredito-syphilitic physiognomy." In a certain number of cases a characteristic dwarfed, notched, dental malformation, will give valuable aid. It is only in the permanent set that any peculiarity is noticed; the milk teeth are liable to decay, but are not pegged, or notched. It is the *upper central incisors* which are the *test-teeth*. Even in grown-up persons, whose incisors are so much worn that the notch is obliterated, the tooth has still a diagnostic form, which Mr. Dixon likens to that of a *screw-driver*, being wide at the neck, and narrow at its cutting edge; its lateral edges are also bluntly rounded off. The complexion is usually pale, or of leaden

FIG. 4.



hue; and though the taint may dwarf the body, in most cases the general growth is not hindered. Mr. Hutchinson has met with several instances of an arrest of sexual development, and Lancereaux with one. A form of phagedenic lupus has been observed; and deafness and amaurosis from nerve or cerebral disease are both far more common in the inherited form of the disease than in that which is acquired, and are usually symmetrical. "As a rule all syphilitic symptoms in the inherited disease are



it often escapes notice, from the absence of the sclerotic zone, the small amount of local symptoms it causes, coupled with the fact that infants usually keep their eyes shut. In 23 cases collected by him, the mean age of the infant was nine months and a half; the oldest was sixteen months, and the youngest six weeks; five were males, and sixteen females, and in two the sex is not given. In 11 cases both eyes were affected. The red sclerotic zone, unfailing in adult iritis, is either wanting or scarcely traceable in the syphilitic form in the infant. The lymph is sometimes scattered over the iris in small isolated granules, but more frequently flows down to the bottom of the anterior chamber, either presenting the appearance known as *hypopyon*, or massed together in a more solid nodular form. The iris becomes dusky, the pupil irregular, and sometimes clouded by the turbidity of the aqueous humor.

The *choroid* is occasionally implicated in hereditary syphilis at about the same period of life as the cornea. The ophthalmoscope shows in such cases the presence of whitish spots on its surface, slightly raised, and covered by the retinal vessels; or the appearance of cicatrices, apparently due to the absorption of these deposits. The retina is congested, and obscured by inflammation of the membrane of the vitreous body. In the first stage, that of exudation, there is lessened vision; in the second the sight improves, the spots becoming defined; the third is that of absorption. In 14 cases reported by Hutchinson, in 10 there was choroiditis (?), in 2 retinal deposits, in 1 inflammatory opacity in the vitreous body, and in 5 opacities in the crystalline lens. In 6 out of 10, the children were the eldest born living.

The stages and symptoms of Inherited Syphilis are clearly presented in the subjoined phenomenal table by Mr. Hutchinson:

**PRIMARY STAGE.**—The infant usually remains without symptoms for from one week to three months.

This stage has been passed through by one or both of the sufferer's parents within from a few months to twenty years of the infant's birth. The infant is usually free from all symptoms at the time of birth.

**SECONDARY STAGE.**—Constitutional or exanthematic.

From the age of two to four weeks to the end of the first year.

This stage is essentially transitory, and will disappear without treatment, if the child lives.

Inflammation of nasal mucous membrane causing "snuffles."

A symmetrical and usually copious eruption on the skin. Wasting; fretfulness; a peculiar odor; a withered senile aspect; inflammation of the mouth and condylomata at anus; iritis, usually symmetrical; arachnitis, and slight effusion; disease of liver (rare); nodes (very rare). The eruptions which occur differ from those of the acquired disease, chiefly in being more moist, and in preferring the thighs and genitals. These differences may in part be due to peculiarities in the skin of young infants, and to the constant irritation from urine to which the nates are liable. Dry scaly rashes are rare. Iritis is much less frequent than in the adult, but just as well characterized when it does occur.

This stage often proves fatal.

**INTERMEDIATE STAGE.**—Stage of latency.

The patient will probably be wholly free from active symptoms, but will show





anatomical character, when the disease is not too rapidly fatal to allow of these pathological features to become developed, as in *yellow fever, typhoid fever, plague, cholera*. These maladies run an acute and rapid course; they are more or less pyrexial; and, in the majority of instances, the fever which accompanies them has a fixed duration. The greater number of them are contagious, or capable of being propagated from person to person, under certain conditions not yet well understood; and, lastly, all of them are produced by an extrinsic poison, either of a *miasmatic* or specifically contaminating nature, as in those of the first order, or by the implanting of a *specific virus*, as in those of the second order.

The *specificity*, so to speak, of these diseases consists in certain characters which distinguish each of them from any other disease, and in the constancy by which, from time immemorial, such characters have continued to distinguish them. Although medical opinions regarding their pathology may change, yet the essential characters of these "*acute specific diseases*" are not known to change. Each of these diseases observes a constancy and regularity of plan in the construction and development of its morbid processes (PAGET). Each of them has some essential character or characteristics by which they are severally distinguishable. The course of the febrile phenomena is found to be distinctive, the duration of the febrile state not less so, as well as the anatomical signs which distinguish the local lesions, the development of which is concurrent with the general or constitutional phenomena; and of all truths relating to the phenomena of disease, the most important are those which relate to the order of their succession. *Specificity* cannot be denied to those diseases in which, during their natural course, we find that every phenomenon is related (in a uniform manner, so far as *exact* investigation has extended) to certain phenomena that coexist with it, and to others that have preceded and will follow it. When it is found that a series of phenomena occur in (thousands, millions)  $x$  number of instances in the same order, within similarly uniform periods of time, and altogether with so much regularity that those who are instructed, on visiting a patient for the first time, can not only affirm what has gone before, but may predict what is to come after (the highest achievement of science)—it is impossible to avoid concluding that such an invariable sequence has as constant a cause. This conclusion flows from the very constitution of our nature, and is inevitable; and on our knowledge of the facts relating to such order of succession is founded every reasonable anticipation of future events, and whatever power we possess of influencing those phenomena in the management of the disease, to the advantage of our patients and the community at large. When it is found, moreover, that there are many series of these phenomena, which may be called A, B, C, D, &c., occurring in different persons, and at different times, all perfectly distinguishable, and never by any chance capable of being confounded by a properly trained person, it is impossible to avoid concluding that the causes of A, B, C, D, &c., are not identical, and must be in fact dissimilar. Moreover, mere *uniformity* in the



With regard to their causes, therefore, each of them appears to be produced by some distinct morbid agent—some morbid poison—a poison or virus which is capable of being multiplied in the body during the development of the particular disease. In this respect they are capable of self-augmentation (PAGET). No evident fresh cause is applied, and yet the disease increases (e. g., *syphilis*, *small-pox*, *vaccinia*, *glanders*, *hydrophobia*, and *malignant pustule*). The theory of each of them, expressed in the most general terms, is, that each of them depends upon a definite specific virus, which induces a morbid condition of the blood; and that, during the development and course of the disease, the system endeavors to discharge or transform in some way the peculiar morbid agents which have given rise to the symptoms, or which have multiplied in the body during the course of the affection. The whole blood then seems to be diseased, and nearly every function and sensation in the frame is impaired or disturbed from the state of health, before any local lesion is developed. Sometimes, indeed, the severest constitutional disturbances of a specific kind may coexist with the smallest local development of any specific lesion (PAGET); and Dr. Robert Williams has justly observed, and numerous examples have been noticed, in which “it may be laid down as a general law, that when a morbid poison acts with its greatest intensity, and produces its severest forms of disease, fewer traces of organic alterations of structure will be found than when the disorder has been of a milder character. Time, duration, or *chronicity*, is a peculiarly important and characteristic element in the nature of these diseases. They run a definite course; and we know of no specific remedy which will at once effect a cure and prove an antidote to the poison. The nearest approach to an antidote is that of quinine in the malarious fevers. They have all—(1.) A more or less defined period of incu-

---

And the statistical return to which I have already often referred (Parliamentary Paper, 1864, No. 12) contains another very striking illustration of the same sort of thing: England has 627 registration districts. During the ten years 1851–60, scarlatina, small-pox, and measles were (as usual) prevailing more or less throughout the country, producing among children under five years of age an average annual mortality of 802 per 100,000; i. e., by scarlatina 419, by small-pox 103, and by measles 280. In 626 of the registration districts there were deaths (and, for the most part, in not inconsiderable quantity) from one or more of those causes; not quite invariably from all of them; for forty-three of the 626 (thanks, no doubt, to vaccination) had not any death by small-pox, and among the forty-three districts which thus escaped mortality by small-pox, there was one which also had not even a single death by measles; but, with these exceptions, all the 626 districts had deaths from the three diseases—deaths by measles, deaths by small-pox, deaths by scarlatina. But the 627th district had an entire escape. In all the ten years it had not a single death by measles, nor a single death by small-pox, nor a single death by scarlet fever. And why? Not because of its general sanitary merits, for it had an average amount of other evidence of unhealthiness. Doubtless, the reason of its escape was that it was insular. It was the *district of the Scilly Isles*; to which it was most improbable that any febrile contagion should come from without. And its escape is an approximative proof that, at least for those ten years, no contagium of measles, nor any contagium of scarlet fever, nor any contagium of small-pox had arisen spontaneously within its limits. I may add that there were only seven districts of England in which no death from diphtheria occurred, and that, of those seven districts, the district of the Scilly Isles was one. Still, to say that a disease is contagious is not to say that it may not arise without contagion” (SIMON, l. c.).



that to some extent the contagious pus may retain its activity when dry enough to float as dust in the air. In some forms of *milzbrand* (including, probably, the so-called 'malignant pustule,' which is the best known human form of the disease) the highly virulent fluids can, it is alleged, infect by soakage through the cuticle. In diphtheria the characteristic exudation is capable of infecting by contact; and though often the disease is communicated from person to person without any manifest transplantation of matter, it may be that in such cases particles of the decomposed false membrane are conveyed as a volatile *contagium*. Cholera and typhoid fever send forth their respective *contagia* for the most part, if not exclusively, as matter dissolved or suspended in the evacuations which pass from the patient's bowels; and probably these evacuations (which, at least in cholera, gradually develop their full infective force after their discharge from the body) can under some circumstances bring into similar contagious fermentation the excrement with which they are mingled in privies, drains, and cesspools, and can thus convert the effluvia and leakage from such sources into means of extensive secondary infection of air and water. The volatile *contagium* of whooping-cough is probably disengaged in large quantities by the air-passages, and as it forms, is sent forth with the breath. In typhus, small-pox, measles, and scarlatina the diffusion of volatile *contagium* occurs to a vast amount, probably with all exhalations from the body; and in addition to this, contagium, more or less fixed, collects abundantly about the patient's person and bedding; and, in a far less degree, something of the same sort probably occurs in erysipelas" (SIMON, *l. c.*). As regards the spread or modes of propagation of these diseases, each of them "has its own laws of communicability,—laws which must be properly understood if the danger of contagion is to be guarded against. The communication of some diseases (of *scabies*, for instance, and *favus*) is not by any true product of the human body, but consists in the migration of parasites, or germs of parasites, animal or vegetable, from one person's body to another,—a migration which of course the recipient may to any extent facilitate by dirty personal habits, and which, as regards some parasitic diseases, can scarcely be conceived to occur otherwise than in consequence of such habits" (SIMON). "The communication of the diseases that have been now considered takes place by that process which is distinctly called zymotic:\* in the

---

\* "Some of these expressions," says Mr. Simon, "are meant to hesitate between two particular assertions. In this respect they correspond to the uncertainty which at present prevails as to the exact nature of some or all morbid ferments. A few years ago it might have seemed permissible to describe without reserve the contagion of the zymotic diseases, as but some changing organic material of the first affected body. At present, however, reserve on that point is necessary. That the power of contagiousness is associated with such changing organic material is certain; but whether the power be *proper to the material*, or be only *contingently* its attribute, seems to require further investigation. The recent very interesting experiments of Professor Shroeder in Germany, and of M. Pasteur in France (published respectively in Wöhler and Liebig's *Annalen der Chemie*, and in the *Comptes Rendus de l'Académie des Sciences*), aim at proving, most extensively, an essential dependence of specific fermentatory and putrefactive changes on the presence, in each case respectively, of some characteristic molecular living thing; and they give it to be



**Effects of Food on the Animal Economy.**—The great fact which recent chemical and physiological investigations have established may be expressed thus: “*That the various alimentary substances made use of by man and animals contain at least four classes of constituents, each of which performs its own assigned function in the living animal economy. If the substance contains nitrogen, it seems most fitted for the nourishment of tissue, and has been called plastic or nitrogenous; if it is deficient in nitrogen, and has an excess of carbon or hydrogen, it appears to undergo combustion in the body, and is called a non-nitrogenous or a respiratory element of food (hydro-carbons); if it is fatty in its nature, it performs the double duty of maintaining animal warmth, and of assisting in the assimilation of nitrogenous compounds; and, lastly, if it is saline in its quality, it goes to build up the solid textures of the animal frame, and aids the important work of carrying new materials into the system, and old or effete matter out of it*” (LETHEBY). Man and animals cannot maintain health if their food does not contain all of these constituents; and common instinct, with experience, tells us that these classes must be associated in due proportions, under a variety of modifying circumstances. There are undoubted *habits* of feeding which, while they appear to be dictated by common instinct, are also sanctioned by science. For example, white meat being deficient in fat, bacon is eaten with veal and with fowl; melted butter is used with fish; eggs and butter are mixed with sago, tapioca, and rice; cheese is eaten with maccaroni; salads and vinegar are eaten with cold or salted meat; a vegetable is mixed with an animal diet; bread is eaten with butter, bacon with greens, pork with pease pudding, and so on. Old habits and instincts not only declare that these combinations are compatible, but Science informs us now why such combinations are demanded for the maintenance of health; and when they cannot be obtained, health is endangered, the constitution is gradually altered, temperament is modified, life is shortened, families extinguished, armies are swept from their encampments, and races of men from the face of the earth.

The experience of Dr. Christison (who has paid great attention to this subject for the last twenty-four years) has shown,—(1.) That the most successful dietaries for bodies of men, deduced from practical observation, contain carboniferous and nitrogenous food in proportion of about three of the former to one of the latter by weight. (2.) That while *nitrogenous* may replace *carboniferous* food for supporting respiration, *though at a great loss, carboniferous* food (without nitrogen) cannot replace *nitrogenous* food for repairing textural waste. (3.) The daily amount of nutritive principles of both sets must increase with exercise and exposure, otherwise the body quickly loses weight, and ere long becomes diseased. If the above proportion between the two sets be maintained, the weight of real nutriment per day varies, for adults at an active age, between seventeen and thirty-six ounces; the former being enough for prisoners confined for short terms, the latter being required for keeping up the athletic constitution, or that which is capable of great continuous muscular efforts. (4.) Dietaries ought never to





and carboniferous food the proportions come out differently. One part of nitrogenous food to from 3 to 6 parts of carboniferous (1 to 4 being the mean) forms the usual proportion in apparently all nations (PARKES).

The histogenetic nature of food must be determined by direct physiological investigation, which should show comparatively the different influence of aliments upon the metamorphosis of matter in the essential animal tissues.

Professor Panum, of the University of Copenhagen, recently instituted a series of experiments to ascertain *the degree of accuracy with which it is possible*, by quantitative determinations of urea, *to discover how much albumen an individual can actually appropriate, digest, and decompose, from an indefinite quantity of food, consisting solely of albuminous matter and water*. His experiments were made on a dog, and he chose the purest albuminate which can be produced in the pure state,—the gluten of wheaten meal. The proportion between the dry albuminate taken and the urea produced was strikingly constant, being, in full feeding, 1 : 4.35 ; in medium feeding, 1 : 3.58 ; and in slight feeding, 1 : 2.81.

Professor Panum also conducted a series of experiments with a view to the solution of the practical question, *whether the production of urea can serve also as a measure of the histogenetic nutritive value of such foods as along with albuminates contain carbo-hydrates or fat in any considerable quantity?* It would appear upon the whole that 1550 grains of starch with 500 grains of butter diminished the production of urea tolerably equally by about 31 grains, corresponding to about 172 grains of dry albuminous matter.]\*

Approximately, it may be concluded that a full-grown man of average weight (140 to 150 lbs.) and height (5 feet 7 inches) requires *one-twentieth* part of his weight in food during the twenty-four hours ; that is, *seven or seven and a half* pounds of food, including solids and liquids ; *one to one and a half* pounds (16 to 24 ounces) being solids, the rest water (PARKES).

On an average, it is found that a man requires *four or five* ounces of chemically dry NITROGENOUS food daily : that in a state of rest he will require *three and a half* ounces ; under a state of considerable exertion *five and a half* ounces ; and under extraordinary exertion he may require as much as *six and a half*, or even *seven* ounces of dry nitrogenous aliment daily. The quantity of HYDRO-CARBONIFEROUS aliment required to keep a man in health cannot be less than from *fourteen and a half* to *fifteen* ounces in twenty-four hours ; and even *nineteen* to *twenty-two* ounces under great exertions. The amount of FATTY MATTERS ought to equal about half the quantity of the *nitrogenous* aliment. But in a state of rest about *one* ounce in the twenty-four hours will be sufficient ; while under great exertion *two and a half* ounces may be required daily. The amount of WATER required varies from *seventy* to *one hundred and thirty* ounces ; and the SALTS supplied in the food should amount to from *half an*

---

\* [For much late, interesting, and valuable matter on the constituents of food and its relation to animal heat, and muscular work, consult *On the Food of Man in Relation to his Useful Work*. By Lyon Playfair, C.B., F.R.S. Edinburgh, 1865. *Review of Panum on Food*, Br. & For. Med. Chir. Rev., July, 1866. Dr. F. Donders *On the Constituents of Food*, Dublin Med. Journal, February and May, 1866.]

ounce to an ounce daily, consisting of *chloride of sodium*, chloride of potassium, salts of *lime* and *magnesia*, carbonates, citrates, lactates, and acetates (PARKES).

TABLE I.—NUTRITIVE VALUE OF FOODS IN 100 PARTS (PARKES).

	Water.	Nitro- genous Substances.	Fat.	Carbo-hydrates, Starch, and Sugar.
Meat without Bone,* . . . . .	74	16	9	...
Fat of Meat,† . . . . .	68	14	14	...
Bread of average quality (White Wheaten), . . . . . }	40	8	1.4	51
Starch, . . . . .	...	...	...	100
Fat, . . . . .	...	...	100	240 of Starch.
Pease, . . . . .	15	24.4	2.6	49
Potatoes, . . . . .	74	1.5	0.6	29
Rice, . . . . .	10	3.8	0.8	85.2
Milk, . . . . .	87	4.3	8.7	5.2
Maize (after Pozziate), . . . . .	13.5	9.9	6.7	64.5
“ (Von Bilra), . . . . .	10.6	15.09	8.8	67.46

\* The mean of usual statements.  
† Calculated by Dr. Parkes from the statements of Lawes and Gilbert.

TABLE II.—DIETARIES AND THEIR NUTRITIVE VALUES (LETHEBY).

Diet.	WEEKLY CONSUMPTION IN OUNCES.							DAILY DITTO.		
	Bread or Biscuit.	Meat.	Potato.	Meal, &c.	Milk.	Cheese.	Butter.	Car- bonif- erous.	Nitro- genous.	Total solid Nutri- ment.
Physiological, . . . . .	140	84	..	..	..	..	3.5	12.7	4.0	16.7
Prison Punishment, E. County and Borough Jails—	112	..	..	..	..	..	..	8.2	1.4	9.6
Under 7 days, . . . . .	121	..	..	23	39.5	..	..	12.4	2.2	14.6
Not hard labor, . . . . .	172	7.8	3.2	22.8	15.4	3.5	..	15.7	3.1	18.8
Hard labor, . . . . .	168	14.6	63.4	27.2	41.6	1.5	..	18.2	3.5	21.7
Scotch Prisons—										
Under 3 days, . . . . .	112	..	..	28	..	..	..	11.2	1.9	13.1
Not hard labor, . . . . .	30	7.5	152	73	175	..	..	19.0	3.4	22.4
Hard labor, . . . . .	76	10	176	100	175	..	..	27.0	4.5	31.5
Irish Prisons—										
Under 1 month, . . . . .	56	..	192	70	70	..	..	19.5	2.9	22.4
Not hard labor, . . . . .	56	..	192	60	170	..	..	20.5	3.4	23.9
Hard labor, . . . . .	64	..	219	70.5	170	..	..	22.0	3.6	25.6
Military Prisons—										
Under 84 days, . . . . .	56	..	..	119	210	..	..	22.2	3.8	26.0
Over 84 days, . . . . .	56	..	..	168	210	..	..	27.8	4.7	32.5
Destitute Debtors, . . . . .	156	16	52	22	21	..	..	16.3	3.1	19.4
Convict Prisons, . . . . .	161	36	112	12	12.8	..	..	18.4	3.6	22.0
Unions (Adults), . . . . .	112	15.5	51	17	34	5.2	1.1	14.2	2.8	17.0
Unions (Children), . . . . .	90	14	32	..	105	..	3.5	11.1	2.3	13.4
Lunatic Asylums, . . . . .	114	23	66	16	14	7	1.3*	13.2	4.0	17.2
Public Hospitals, . . . . .	93	52	56	14	7	..	3.2	12.1	3.5	15.6
Army—										
Crimea, . . . . .	112†	112	..	..	..	..	..	14.5	4.8	19.3
Home, . . . . .	168	84	112	..	..	..	..	19.4	4.8	24.2
Madras, . . . . .	112	112	56	4*	..	..	..	16.5	4.9	21.4
Bombay, . . . . .	140	112	56	56*	..	..	..	22.2	5.6	27.8
Field (India), . . . . .	168	..	..	168*	..	..	..	30.7	3.8	34.5
Navy, . . . . .	112†	112	56	..	..	..	..	17.7	5.0	22.7
† Navigator (Crimea), . . . . .	140	140	..	28	..	..	..	17.8	6.2	24.0
† Navigator (Home), . . . . .	320	96	64	..	..	12	4	18.6	7.7	26.3
Berwickshire Laborer, . . . . .	122	..	..	224	224	..	..	37.1	7.0	44.1
Yorkshire Laborer, . . . . .	280	126	28	..	210	..	49	42.2	8.8	51.0

In this table only the most important articles of diet are mentioned, although the others, excepting beer, spirits, tea, and coffee, are calculated in the daily consumption. (\*) are rations of rice, and (†) rations of biscuit. Gruel is calculated at the rate of two ounces meal per pint.  
‡ A name given to those laborers who are employed in excavating, and such-like laborious work, chiefly connected with the construction of railways. They are also sometimes called “navvies.”

To determine by calculation the amount of these different aliments, and therefore the nutritive value of a given diet, the following scale is given (Table I). It shows the mean amount of water, nitrogenous substances, fat, and carbo-hydrates—starch and sugar—which ought to be contained in 100 parts of each of the substances in common use mentioned in Table I.\*

The calamities which befell our soldiers in the Crimea (in 1854) show that the dietaries of working men cannot be safely reduced below the physiological standard; and, in the words of Dr. Christison, "any person conversant with the science of the present subject could have foretold, as a certain consequence, sooner or later, of their dietary, that the British would fall into the calamitous state of health which befell them in the Crimea."

The preceding very interesting tables (II and III) of dietaries and their nutritive values, and of the nutritive values of foods, by Dr. Letheby, of London, are given here to show the actual proportions in which various substances used as food are associated in the several public dietaries of the country, and as a guide to the student of medicine when, as a practitioner, it may often be his lot to devise and construct scales of diet suited to various conditions of existence.

TABLE III.—NUTRITIVE VALUE OF FOODS (LETHEBY).

SUBSTANCES, 100 PARTS.	Water.	Fibrine, Albumen, &c.	Starch, Sugar, &c.	Nitro- genous.	Total Nutri- ment.
Human Milk, . . . . .	89	3.5	4.2	8.5	14.9
Cow's Milk, . . . . .	86	4.5	5.0	4.5	19.3
Skimmed Milk, . . . . .	87	4.5	5.0	4.5	18.0
Buttermilk, . . . . .	87	4.5	5.0	4.5	10.5
Beef and Mutton, . . . . .	73	19.0	..	19.0	31.0
Veal, . . . . .	77	19.0	..	19.0	31.4
Poultry, . . . . .	74	21.0	..	21.0	38.2
Bacon, . . . . .	30	0.8	..	0.8	106.3
Cheese (Cheddar), . . . . .	36	22.9	..	29.0	101.0
" (Skimmed), . . . . .	44	45.0	..	45.0	69.4
Butter, . . . . .	15	..	..	..	190.0
Eggs, . . . . .	74	14.0	..	14.0	39.0
White of Egg, . . . . .	78	20.0	..	20.0	30.0
Yolk of Egg, . . . . .	42	16.0	..	16.0	38.0
White Fish, . . . . .	79	19.0	..	19.0	31.4
Salmon, . . . . .	78	17.0	..	17.0	36.6
Eel, . . . . .	90	10.0	..	10.0	29.3
Wheat Flour, . . . . .	15	11.0	70.0	11.0	85.8
Barley-meal, . . . . .	15	10.0	70.0	10.0	85.8
Oat-meal, . . . . .	15	12.0	62.0	12.0	88.4
Rye-meal, . . . . .	15	9.0	66.0	9.0	79.6
Indian-meal, . . . . .	14	9.0	65.0	9.0	83.2
Rice, . . . . .	14	7.0	76.0	7.0	83.7
Haricot, . . . . .	19	23.0	45.0	23.0	75.2
Peas, . . . . .	13	22.0	55.0	22.0	84.9
Beans, . . . . .	14	24.0	44.0	24.0	71.4
Lentils, . . . . .	14	29.0	44.0	29.0	76.6
Wheat Bread, . . . . .	44	9.0	49.0	9.0	60.4
Rye Bread, . . . . .	45	5.0	46.0	5.3	53.7
Potatoes, . . . . .	74	2.0	23.0	2.0	26.5
Green Vegetables, . . . . .	65	2.0	4.0	2.0	7.0
Arrow-roots, . . . . .	18	..	82.0	..	32.0

In this table the carboniferous matter is calculated as starch; 10 of fat being equal to 24 of starch.

\* This table is used by Dr. Parkes in the Laboratory of the Army Medical School, and he kindly permits me to give it here.



“Experience has shown,” says Dr. Letheby, “that there are certain articles of food which are not particularly nourishing in themselves, but which serve some very important purposes in the animal economy. This is the case with tea and coffee: in fact, the use of a vegetable infusion, containing astringent matter and an active principle rich in nitrogen, has been almost universal among mankind from the earliest times.”

“The physiological action of these beverages appears to be of a somewhat singular kind; for while they excite the brain, they calm the nervous system generally, and though they produce a state of wakefulness and activity, yet they also induce a species of languor and repose. Lehmann has ascertained by experiment that coffee greatly diminishes the wear and tear of the system; it oils the machinery, as it were, and checks the waste of friction; for those who use it find that during active exercise the destruction of tissue is prevented, and that there is less demand for food; in fact, with a maximum of work to perform, and a minimum of food to accomplish it, he will best sustain his vital power who resorts now and then to a cup of tea or coffee. Hence its value as a means of economizing food, and hence its importance to the poor laboring man.” In many of our large merchant-ships the crews are engaged on the condition that coffee shall take the place of grog; and those captains who are careful of the health of the men, give them warm coffee before or after they have been aloft in cold and stormy weather—a practice which cannot be too much overvalued.

**Effects of Overfeeding.**—Too much respiratory food favors the development of fat, and checks the proper nutrition of the muscular tissues; hence it is that rice-feeders and potato-eaters, and those who indulge in fermented liquors, are often bloated in their appearance, become extremely fat, and are not capable of prolonged exertion. The brewer’s drayman is a bad subject for the wards of an hospital; for though he usually has all the appearance of a man possessed of great muscular strength and vital endurance, yet he is not so in reality, for the muscular tissues have been encroached upon by fat, and the general power has been weakened by an undue influence of the respiratory element. Most of the animals in our menageries, from a too liberal allowance of respiratory food, die from fatty degeneration. Accumulation of the nitrogenous elements in the blood is often also a prolific source of disease, and their non-elimination (as shown in previous pages) is conducive to the propagation and development of many *miasmatic* diseases. Attention has now been drawn to the influence of such a condition in establishing the characteristic diseases of overfed convicts (LETHEBY, THOMPSON).

**[Corpulence.]**—This is one of the most distressing results from the effects of overfeeding, or from perverted assimilation. The adipose tissue ought to form about one-twentieth part of the weight of man, and one-sixteenth of woman. Corpulence is to be considered a disease when “it renders persons, from a difficult respiration, uneasy in themselves, and, from the inability to exercise, unfit for discharging the duties of life to



ing long confinement from surgical operations, or chronic diseases, in which general nutrition is not affected.

**Prognosis.**—When it shows itself in early infancy and goes on increasing with age, it is incurable. In childhood and early puberty it may be checked. The later it is developed the easier it is controlled. Excessive corpulence does not betoken long life. Daniel Lambert (740 pounds), died in his fortieth year; and Bright, of Malden (616 pounds), only lived twenty-eight years.\* Dancel mentions the case of a man (640 pounds), who died suddenly, suffocated with fat, at about thirty years of age. There is always in such persons a tendency to fatty degenerations of the organs.

**Treatment.**—By modification of the diet, we are measurably able to hinder the tendency to the formation of fat in the system, and even, to a certain extent, to lessen present amount, without otherwise doing harm to the sufferer; but the treatment must be managed with care and discretion. A neglect of the due adjustment between the fats and albuminates in the food is often followed by serious disorder of the nervous system, feeble digestion, obstinate constipation, gouty symptoms (from accumulation of the lithates), general sluggishness, and, in time, evidences of some degree of malnutrition, as the writer has several times had occasion to notice in those undergoing the so-called "Banting process." It should be borne in mind, too, that the capability for muscular work does not depend alone on the degree of development of the sarcous elements, but also on nervous action, and that fat is a nerve-nourisher, and its absence cannot fail to be felt in the nervous system; the limited endurance of prize-fighters, whose nerve-tissue is starved during "training," is well known. There exists, also, a certain relation between heat produced and muscular work, and both are derived from the chemical energy of non-nitrogenous as well as nitrogenous matters (DONDEERS).† The drift in man everywhere is toward mixed food, and with its use the best physical state is reached. Experience shows that the deprivation of the starches can be borne for a long time, if fat be given; but the deprivation of fat is ill-borne, even if starches be given. In many disorders of malnutrition, fat is found efficient as among the remedies, meeting some indication, and supplying some want. The salts, too, especially those which form carbonates in the system, are necessary to the integrity of the molecular currents, and form parts of nearly all the tissues; they exist chiefly in fresh vegetables. Hence the necessity of watchfulness and prudence in regulating the food in corpulency. When living exclusively on animal food, upon the setting in of any unpleasant symptoms, the rigorous nitrogenous diet must be modified. It should be remembered too, that mental workers do not bear abstinence, and particularly deprivation of the fats and carbohydrates, so well as body workers.

Although all corpulent persons are not huge feeders, a large number of them are; they must "leave gormandizing." The oleaginous, starchy, and saccharine articles of food should be sparingly taken, or, for a while, abstained from, under the rules already laid down. The meals should be light, and eaten at comparatively brief intervals—shortening the process

---

\* [Shakspeare, who has given with his accustomed accuracy all the mental and physical characteristics of corpulence in that "candle mine," the "greasy knight," allows "his age some fifty, or inclining to threescore." (1st Part of King Henry 4th, Act II, Scene IV.)]

† [*On the Constituents of Food and their Relation to Muscular Work and Animal Heat.* By F. C. Donders, M.D. Translated by W. D. Moore, M.D., Dublin Quar. Jour. of Med. Science, February and May, 1866 ]



of digestion, and lessening the time any mass of food shall lie in the stomach. Liquids, and especially water, should be taken in small quantities, and at the end of the meal. The amount of alcohol permitted should be very small: not that it is directly a fat-former, but, as Böcker's experiments have shown, it diminishes the excretion of carbon. Malt liquors and champagne should be strictly prohibited. The light wines of France and Germany, mixed with Vichy water, may be allowed. If tea is insisted upon, it must be used without milk or sugar: a slice of lemon, Russian fashion, may be thrown on top. Turkish baths may be often prescribed with advantage, if they are followed by a feeling of "stiffness," and not of lassitude, and there is no reason to suspect fatty degeneration of any internal organ. Warm, alkaline, and salt baths, are of use by their action on the skin, and should be aided by daily friction over the whole body. During the use of the nitrogenous diet, Dr. T. K. Chambers recommends full doses of the *liquor potassæ* daily; and Dancel the *bicarbonate of soda*. Five to ten grains of tartaric acid are dissolved in two-thirds of a tumbler of cold water, and from thirty grains to one drachm of bicarbonate of soda are added: two-thirds of the solution to be drunk during effervescence. (*Traité Théorique et Pratique de l'Obésité (trop grand embonpoint)*, etc. Par F. Dancel. Paris, 1863.) The writer has given with advantage, as he thinks, and with a view to better tolerance of the nitrogenous diet, both phosphoric and citric acids, and lemon-juice. Bromide of ammonium has been recommended. Active exercise, in the open air and in sunlight, must not be neglected. Dancel claims that *scammony* possesses the special property of bringing on greasy dejections, fat, after its use for awhile, coming away in the stools, and sometimes in lumps. He associates, in his treatment of corpulence, along with the received dietetic rules, doses of the *tincture of pure scammony*, given before breakfast, every second or third day, in a capsule: he asserts that it is well borne by the stomach. The resin of scammony being less irritating, might be advantageously substituted. Local supporters, in the form of body and breast belts, may be worn with comfort.

Mr. Banting, the court undertaker, whose pamphlet *On Corpulence* is so widely known, was placed under the following dietary by Mr. William Harvey, a London surgeon, who, in prescribing it, followed out the already published views of Dr. Chambers and of Dancel.

*Breakfast*—Four or five ounces of beef, mutton, kidneys, boiled fish, bacon, or cold meat of any kind (except pork), a large cup of tea (without milk or sugar), and one ounce of dry toast. *Dinner*—Five or six ounces of any fish (except salmon or eels), any meat (except pork), any vegetables (except potatoes or rice), one ounce of dry toast, fruit out of any pudding, any kind of poultry or game, and two or three glasses of good claret, sherry, or Madeira (champagne, port, or beer forbidden). *Tea*—Two or three ounces of fresh fruit, or a rusk or two, and a cup of tea without milk or sugar. The tea may be very much enjoyed when taken in the Russian fashion—*i. e.*, with a thick slice of lemon floating on the top instead of milk. *Supper*—Three or four ounces of meat or fish, similar to dinner, with a glass or two of claret. *Nightcap*, if required, a tumbler of grog (gin, whisky, or brandy, without sugar), or a glass or two of claret or sherry.

The quantities of the different articles specified in this liberal diet-roll, Mr. Banting states, must be left to the natural appetite, but for himself he took at *breakfast* six ounces of solid and eight of liquid food; at *dinner*, eight ounces of solid and eight of liquid; at *tea*, three ounces of

solid and eight of liquid; at *supper*, four ounces of solid and six of liquid; and the *nightcap* he introduces to show that it is not injurious; whilst, for the encouragement of smokers, it may be mentioned that tobacco is allowable.

When Mr. Banting began this treatment in August, 1862, he weighed 202 lbs., and after a year's perseverance in it, in September, 1863, he had lost 46 lbs., and had reduced his girth 12½ inches.]

**Effects of Deficient Food.**—"A deficiency of food, especially of the nitrogenous part, quickly leads to the breaking up of the animal frame. Plague, pestilence, and famine are associated with each other in the public mind, and the records of every country show how closely they are related. The medical history of Ireland is remarkable for the illustrations of how much mischief may be occasioned by a general deficiency of food. Always the habitat of fever, it every now and then becomes the very hotbed of its propagation and development. Let there be but a small failure in the usual imperfect supply of food, and the lurking seeds of pestilence are ready to burst into frightful activity. The famine of the present century is but a too forcible illustration of this. It fostered epidemics which had not been witnessed in this generation, and gave rise to scenes of devastation and misery which are not surpassed by the most appalling epidemic of the Middle Ages. The principal form of the scourge was known as the contagious famine fever (typhus), and it spread, not merely from end to end of the country in which it had originated, but, breaking through all boundaries, it crossed the broad ocean, and made itself painfully manifest in localities where it was previously unknown. Thousands fell under the virulence of its action, for wheresoever it came it struck down a seventh of the people, and of those whom it attacked one out of nine perished. Even those who escaped the fatal influence of it were left the miserable victims of scurvy and low fever. Another example, not less striking, of the terrible consequences of what may be truly called famine, was the condition of our troops during the early part of their sojourn in the Crimea, in 1854. With only just enough of food to maintain the integrity of the system at a time of repose, and at ordinary temperatures, they were called upon to make large muscular exertions, and to sustain the warmth of the system, in the midst of severe cold" (LETHEBY).

In cases of very gradual starvation an urgent feeling of hunger is not a prominent symptom, and even when it exists at first, it usually soon diminishes, and is succeeded by a feeling of exhaustion and faintness, and even loathing of food, if abstinence has been long protracted (R. B. HOLLAND). The mental condition connected with poverty may in part account for this deficiency of appetite. A depression produced on the nervous system is very early manifested in the impaired energies of all the vital functions, the weakened conditions of the intellectual faculties and moral feelings, and diminution of the general sensibility. Disturbance of the cerebral functions is at first shown by an unnatural languor, despondency, and listlessness, slowness and hebetude of intellect, with an inability to



Trousseau, he lives on himself. This condition is constantly met with in continued fevers, where strict diet has been enforced for any length of time, cancer of the stomach, cholera infantum, chronic diarrhœa, &c.

The quantitative estimation of the excretion of urea in a starving animal may be regarded as a pretty accurate measure of the decomposition of the essential nitrogenous tissues during inanition. Now a male adult in health excretes only about 460 grains of urea in the twenty-four hours, and if fasting, half the quantity; whilst in continued fever, pyæmia, and even acute pneumonia, the urea may rise to 775 grains, 1240 grains, and 900 or even 1075 grains, respectively, in the twenty-four hours.]

When privations of clothing and lodging are added to insufficient diet, long exertion, insufficient repose, intemperance, and the miseries of poverty, the symptoms already detailed are of the most aggravated kind. "Long before insufficiency of diet is a matter of hygienic concern—long before the physiologist would think of counting the grains of nitrogen and carbon which intervene between life and starvation—the household will have been utterly destitute of material comfort; clothing and fuel will have been even scantier than food; against inclemencies of weather there will have been no adequate protection; dwelling-space will have been stinted to the degree in which over-crowding produces or increases disease; of household utensils and furniture there will have been scarcely any,—even cleanliness will have been costly or difficult; and, if there still be self-respectful endeavors to maintain it, every such endeavor will represent additional pangs of hunger. The home, too, will be where shelter can be cheapest bought,—in quarters where commonly there is least fruit of sanitary supervision, least drainage, least scavenging, least suppression of public nuisances, least, or worst, water supply, and, if in town, least light and air. Such are the sanitary dangers to which poverty is almost certainly exposed, when it is poverty enough to imply scantiness of food" (Simon, *Sixth Report on Public Health*, 1864, p. 14). But a multitude of cases of minor degrees of suffering occur in which the symptoms are less marked than those described. Such cases are indicated by a sallow and dingy appearance of the skin, a soft and flabby feeling of the flesh, more or less emaciation, general debility, feebleness of the circulation, and frequently swelling of the ankles. The stomach becomes disordered, the appetite defective, and digestion impaired. The individual feels languid and desponding, is soon fatigued, incapable of exertion, and has an irresistible desire to fall asleep, from which he is apt to awake suddenly and in a fright. The body is easily chilled, breathlessness and palpitation are experienced after slight exertion, attacks of *vertigo*, *tinnitus aurium*, and transient blindness, are common, and there is a peculiar forlorn and dejected aspect of countenance which is very characteristic. [Aphthæ are of common occurrence in all disorders involving general malnutrition.] This state of things is commonly soon succeeded by some specific disease; though it sometimes continues, with only slight variation, for a very protracted period, until the patient falls by slow degrees into a state of mental as well

as physical incapacity; and, being no longer able to procure any employment, is completely invalided, and applies for medical relief. It may perhaps be thought that these remarks apply to cases of deficient nourishment, which are less frequent; but the experience of those who have practised extensively among the wretched purlieus and miserable abodes which exist in every large metropolitan town can testify to the contrary. I well remember listening to the interesting clinical lectures of Dr. Christison, of Edinburgh, on the cases of *scurvy* which prevailed in that town and its vicinity in 1847, and hearing the melancholy recitals of misery and starvation under which the poor suffered at that time; some under the hard taskmasters of the illegal "truck system,"\* and others from absolute want at home. Among many, of whose cases I have preserved notes, a shoemaker had to support his wife and five children on *eight shillings* a week; and, to feed his children better than himself, he subjected himself to privations which in time developed *scurvy*. His daily diet consisted of *one pennynorth of bread, with tea, but no milk, in the morning—no dinner—and one pennynorth of bread, with tea, and no milk, in the evening*. After existing *three months* on this diet the disease broke out. But, apart from these extreme cases, the instances are innumerable in which deficiency of food acts as a predisposing cause of many diseases. It is now generally known that plethora and symptoms of an opposite state very nearly resemble each other, and a discrimination of these differences is of the greatest importance. While coma is often an attendant on plethora, it is not to be forgotten that it is one of the most severe and fatal signs of exhaustion from defective nutrition: and when it supervenes towards the termination of diseases of exhaustion, and the pulse becomes slower, it often acquires a degree of fulness, and gives an idea of strength, quite at variance with its previous character, and little to have been anticipated from the debilitated state of the system.

But in degrees far short of what is popularly known as starvation or famine, insufficiency of nourishment may bring very hurtful consequences to health. Local defects or local peculiarities of diet may exercise an important influence in determining or coloring particular localizations of disease; and generally it may be said, that in order justly to estimate the sanitary circumstances of a people, sufficient regard must be had to the quantity and quality of the people's meat and drink (Simon, *Report on Public Health*, p. 11, 1864).

The injurious effects produced by improper nutrition require to be studied both in relation to food and drink; and the diseases which belong to this order are *scurvy, purpura, famine fever, alcoholism, and*

---

\* The "*truck system*" became developed chiefly during the formation of our great lines of railway throughout the country. The laborers (navvies) were poor, and came to work without money to buy provisions, and their field of labor was often far removed from any place where food could be bought in quantity. The contractors, their employers, then established provision stores, and in place of paying the men in money, they compelled them to take remuneration for their labor by value received in food. By this method, now declared illegal under all circumstances, the laborers often suffered from a deficient and bad supply of provisions.

probably also *rickets*. The place of *cretinism* and *bronchocele* cannot be said as yet to be definitely determined. According, however, to recent investigations, these diseases are found among people of all habits in the countries where they abound, only where the soils are composed of magnesian limestone rocks, where the waters contain an excess of magnesian salts, in France, Germany, England, Sardinia, America, and India. (See page 202.) Through the water, therefore, as an element of diet, these diseases appear to become developed, and, therefore, in the meantime, they may be arranged among the diseases of this order, some of which now demand a detailed description.

[The effects of continued insufficient alimentation have been graphically described by De Meersman, as observed in Belgium during the famine years of 1846-47 (quoted by Longet in his *Traité de Physiologie*, t. i.). The extreme emaciation of the body, pallid face, and sunken cheeks, the bright eye and dilated pupil, haggard, bewildered look, the weak, tremulous voice, the feeble memory, infirm mind, the slow, uncertain, tottering gait, dry, yellow, parchment-like, and fetid skin, stinking breath, shrunk belly, slow, sighing respiration, small, frequent, and gaseous pulse, are all described with sickening fidelity. But the largest field for the observation of the consequences of gradual continuous starvation was afforded by the Andersonville military prison towards the end of the late war, where thirty thousand men were exposed, within an area of twenty-seven acres, without shelter, and with food insufficient in quantity and quality, to the weather, with all the ills of overcrowding, and were literally, slowly and surely, starved to death. The report of Professor Joseph Jones, of Nashville, Tenn., made to the Surgeon-General of the Confederate Army, on the condition of the prisoners of war, has been well called "the most complete scientific history of inanition ever written, deduced from data which are, and probably always will be, unparalleled in magnitude." This, in a medical point of view, invaluable and instructive report, is published at length in the *Medical Memoirs of the U. S. Sanitary Commission*. (See also *Report of a Commission of Inquiry appointed by the U. S. Sanitary Commission*, Philadelphia, 1864.) ]

---

## CHAPTER X.

### DETAILED DESCRIPTION OF THE DIETIC ORDER OF ZYMOTIC DISEASES.

#### SCURVY.

LATIN Eq., *Scorbutus* ; FRENCH Eq., *Scorbut* ; GERMAN Eq., *Scorbut* ; ITALIAN Eq., *Scorbuto*.

**Definition.**—A chronic morbid state ushered in by debility, lassitude, lowness of spirits, attended by fetor of the breath, sponginess of the gums, which swell by irritation, till they overhang the teeth in palmated excrescences. Livid subcutaneous patches and spots appear upon the skin, of considerable extent, especially on the lower extremities among the roots of the hair. Spontaneous hemorrhages may take place from





in America it is now very little known, and many have never seen the disease; unless they have lived in seaport towns.

[Scurvy has always prevailed in the United States Army to a considerable extent, and under the same circumstances,—a deprivation of fresh vegetables. From 1840 to 1859, inclusive (excluding the years 1847–48—the period of the Mexican war—no reports having been made during that time), there were 4935 cases of scurvy reported and 52 deaths, in an aggregate strength of 187,144 men (*Statistical Report U. S. Army. By authority.* 1860). In the column which marched on the city of Mexico, the men, for some time previous to their landing at Vera Cruz, during the siege, and afterwards, could obtain no vegetables, and the writer is informed, on reliable authority, that on their arrival at Jalapa, although there had been plenty of fresh beef, there was scarcely a man who did not have a scorbutic taint. During the late civil war, scurvy, in some form or another, was generally prevalent in our armies. Though the statistics of the first two years show an extremely small number of cases, “unparalleled in the history of armies,” it is not a true exhibit of the actual prevalence of the disorder. There were reported in the first year, 1328 cases and 9 deaths, and in the second year, 7395 cases and 90 deaths: to which may probably be added 304 cases of purpura and 31 deaths (*Circular No. 6, Surgeon-General's Office, War Dept.,* 1865). It first appeared to any extent in the Army of the Potomac, at Harrison's Landing, Va., in July, 1862, and from that time forward, until the end of the war in 1865, it continued an increasing and formidable disorder. “It occurred in all [the] armies subjected to hardships, especially in the West, and its worst and most fatal manifestation was made after the war had closed, and during the occupancy of the frontier of the Rio Grande by the twenty-fifth Army Corps,” and in consequence of the want of fresh vegetable food (*U. S. Sanitary Commission Memoirs*).

What has been written of scurvy in the British army, during the Crimean war, is equally applicable to our own, during the late war. “The returns convey but a faint conception of the disastrous part which it [scurvy] acted among the troops; for though it comparatively rarely presented itself in well-defined forms, and as an independent affection, yet the prevalence of scorbutic taint was widespread, and in a vast proportion of cases evident indications of it existed as a complication of other diseases, especially fevers and affections of the bowels” (*Medical and Surgical History of the British Army*, 1858).

In the first years of the war scurvy did not prevail amongst the Confederate troops apparently to any extent, for with a monthly mean strength of 160,231 officers and men, and with 1,056,349 cases of sickness and wounds entered upon the field reports in nineteen months, from January, 1862, to July, 1863, only 2203 cases of scurvy are recorded; and in 398,641 cases of sickness and wounds entered upon the hospital reports, there were only 2068 cases. But it progressively increased, with a diminishing commissariat and increased hardships (*J. JONES, in San. Com. Memoirs.*) The same authority, in his report on the Diseases, &c., of the Andersonville prison, states that scurvy, arising from sameness of food and imperfect nutrition, caused, either directly or indirectly, nine-tenths of the deaths amongst the United States army prisoners confined there.]

[**Morbid Anatomy and Pathogeny.**]—The days when scurvy was most prevalent were not those in which many post-mortem examinations were made, and our earliest knowledge of the morbid anatomy of





ness; and their capillaries capable of being injected, like other adventitious membranes—"scorbutic formation" (HEMMELSTIERN). There are serous or fibro-serous effusions beneath the arachnoid, or a reddish fluid, like currant-jelly water, is infiltrated into the subserous areolar tissue. Ecchymosed spots or patches upon the surface of the brain, and occasionally into its substance, particularly at the base, and near the pes hippocampi. Intra-pleural effusions. The lungs may be pale, bloodless, and gorged with serum; or there may be superficial violet stains or marblings on their surface; or wine-colored fluid in the pulmonary vesicles, intercellular passages, and subserous connective tissue. In those cases where special lung symptoms have been present, portions of the pulmonary substance, generally in the lower lobes, are gorged with black blood, and are inelastic, uncrepitant, and impermeable. Again, there may be ecchymosed spots in the interlobular connective tissue; or large, fluctuating grumous masses, in defined, irregular cavities, not lined, may exist in the lung parenchyma. Occasionally there is gangrene of the lung. Ecchymosed spots or patches in the subserous pericardial tissue, on the surface of the heart, and intermuscular connective tissue. Spleen softened. On the mucous and in the submucous tissues of the stomach and bowels, ecchymoses, with often ulcers of the large intestines. (See *Chronic Camp Dysentery*, p. 590, vol. i.)]

The blood appears to be deficient in red particles (BUSK, BECQUEREL, RODIER, ANDRAL, FRICKS), and does not impart a stain to the lining membrane of the heart and great vessels. A fluid or dissolved condition of the blood has also been noticed, appearing often as "a mere gore, not separating into crassamentum and serum, and putrefying soon. It appears to be starved of some essential ingredient." It flows with difficulty from the vein, and, after standing some hours, deposits a thick, muddy sediment, which subsides from a reddish serum; and in the last stage of the disease it becomes quite black (ROUPPE). There does not, however, appear to be any evidence of deficient powers of coagulation in scorbutic blood; and so long ago as 1699, Poupart noted the large coagula found after death in the cavities of the heart. Three analyses of the blood were also made by Mr. Busk, before 1840, in all of which the quantity of fibrine was found to be above its normal standard. Stoeber, in 1845, came to the same conclusion; and so did Andral, on repeating analyses which at first led him to an opposite conclusion. Two facts of great importance, if confirmed, have been observed by Chatin and Bouvier. They have observed that the *albumen* of the blood does not coagulate under a temperature of 74° Cent., or 165.2° Fahr.—that is, from 5° to 8° Fahr. above the normal standard; so that the albumen of the blood undergoes some change in scurvy which increases its solubility. They also observed that the force of cohesion of the *fibrine* was so much lessened that they were unable to isolate it thoroughly from the red corpuscles, and it was this attraction of the fibrine and the red corpuscles which the early writers on scurvy attempted to describe by the terms "*agglutinated blood*," "*viscid and thickened crassamentum*" (Parkes "On the Pathology and Treatment of Scurvy," *Brit. and For. Med.-Chir. Review*, Oct., 1848). Thus all the phenomena of scurvy, and the conditions under which it becomes developed,



of the earliest symptoms of the disease. "Opposed also to this theory," writes Dr. Wood, "are the facts that nitrate of potash has often failed to cure the disease, while it has been cured by the addition of pure citric acid; and that, after failure under treatment with the salts of potash, recovery immediately commenced when the patient was allowed to eat fresh vegetables." Trotter also always held the opinion that the real antiscorbutic principles in fruit and vegetables were the vegetable acids, particularly citric, oxalic, and malic acids; and to test the accuracy of his opinion, he made, in 1800, a series of comparative trials between *lemon-juice* and pure *citric acid*. Both these remedies were furnished to eight or ten ships, and reports were then obtained of their effects. The result was that *citric acid* was found the most efficacious. Sir William Burnett, also, was in the habit of supplying convict ships with *citric acid* and *nitrate of potash*, as well as with *lemon-juice*, in order that comparative trials might be made of the relative value of these remedies; and the official documents bearing on this point were carefully examined by Dr. Parkes, who considered that nothing could be more convincing than the evidence they contained, showing that the efficacy of *citric acid* was clearly proved, while *nitrate of potash* was shown to be inferior in curative power (*On the Pathology and Treatment of Scurvy*, l. c.).\* The astonishing effects of *fresh lemon-juice* have been evidently underrated by Dr. Garrod; and it is more probable that its virtues are due to citric and other acids than to potash. Dr. Aldridge contends for the influence which should be ascribed to a deficiency of phosphorus, sulphur, lime, and the alkalies, in occasioning scurvy. That something may be owing—a part merely—to the causes contended for by Dr. Aldridge is not improbable. But it is unnecessary to pursue this subject any further than very briefly to state, that one of the most evident changes from the healthy condition is seated in the blood, which is altered in composition either by the addition to it of some ingredient or ingredients, or by the absence of something which ought to exist in it; and the deficient ingredient may be one of the ordinary constituents of the blood, or it may be some principle or element entering into their composition. This deficiency is due to the absence of certain articles of diet; and the disease is known by experience to be at once cured by supplying those articles.

**Symptoms.**—The earliest are a change of color of the skin, particularly of the face and eyelids.

[Sometimes the condition of the eye and its appendages is the first and only physical sign of the disorder. The skin around one or both orbits is puffed up into a bruise-colored swelling; the sclerotic conjunctiva is tumid and of a brilliant red color throughout, and elevated about one-eighth of an inch, the cornea appearing at the bottom of a circular well. There is no pain or discharge. It betokens a serious form (BIRD, BELLINGHAM). Breathlessness is often one of the initial symptoms.]

---

\* [Nitrate of potash is inefficacious against scurvy (MURRAY, BRYSON). The Ottawa lumberers living on pork salted with nitre suffer greatly. Dr. J. O. Grant found in one shanty out of 36 of these men 25 affected with scurvy. (*Med. Times and Gaz.*, vol. ii, 1868.)—EDITOR.]



the derm, forming brown spots, which may last for months, the epidermis over these being smooth and shining.

In the *subdermic ecchymomata* the blotches are larger and of variable depth. They may invade the whole limb, but the most common site is the ham, groin above and below Poupert's ligament, the thigh, calf, and pterygo-maxillary region. If they compress superficial veins, they cause œdema and pain. They produce swellings in the flexures of the joints, most often in the ham, or shin, in the pterygo-maxillary region, and bend of the elbow. They are hard, but pit on persistent and firm pressure. When on the shin and circumscribed, they may be mistaken for syphilitic nodes. Resolution is their usual termination, their hue changing from dark purple to green and yellow. When the dark color persists with a greenish-yellow border, it is evidence that the effusion is very thick. Should their termination be by ulceration, a sort of indolent boil forms, with a deep-red base and black summit, with œdema of the adjacent connective tissue. If the surface is galled, there is constant weeping of a sero-sanguinolent fluid; the tumor flattens a little; and a small, dark, ulcerated central point, with wine-red edges, appears: this is a gangrenous mass, which gains little by little, until an ulcer of variable size is established. The most frequent site of these scorbutic ulcers are the calves of the legs, the buttocks, thighs, sacrum, shoulders, arms, and more rarely the chest and abdomen. The *deep-seated extravasations* are, (1) *sub-aponeurotic*; (2) *parenchymal*, into the muscles, kidneys, heart, lungs, &c. These extravasations into the muscle-tissue never end in suppuration, but the tissue becomes atrophied, and undergoes a sort of gelatinous degeneration; resolution is very sluggish. When the effusion is beneath a resisting aponeurosis, as the fascia lata, there is no alteration in the integument immediately over it, but the swelling and discoloration happen at remote points, as the ham, about the knee, &c. The source of these extravasations would seem to be the softened capillary vessels, permitting the leakage of altered blood; there is no apparent solution of continuity except an ulcer exists (MARBY, *loc. cit.*.)]

The tongue is now white, the breath fetid, and the stools generally pale.

[Ptyalism may take place, with swelling of the parotids and submaxillary glands, and soft, swollen, or ulcerated gums and tongue, giving the look of mercurialization (RITCHIE).]

As the disease advances, all these symptoms are aggravated. The loss of physical power increases, the purple spots have a tendency to ulcerate, and the ulcers are distinguished from all others by their putrid fungoid appearance and great tendency to bleed; old sores open, and the callus of broken bones has even been dissolved and their ends separated.

[The mucous membrane of the gums, cheeks, and even pharynx, sphacelates in shreds. The cheek may be destroyed in spots, with constant leakage of a fluid composed of saliva, blood, and gangrenous tissue, exhaling a putrid odor. In such cases a diphtheritic angina is apt to intercur, with œdema of the glottis, and is usually fatal.]

Profuse hemorrhages frequently take place from the mouth, nose, [pharynx], lungs, [genito-urinary organs], or bowels.



damp spells. It begins with slight rigors, followed by feverishness, and accompanied by lancinating pains in one or both sides. There is dyspnoea, and a feeling of constriction in the chest, as if a cord were drawn tightly round it. This condition is commonly caused by intra-pleural effusion of blood, but sometimes blood escapes into the pulmonary tissue; in the latter case, the expectoration becomes dark and sanious, and has a fetor resembling that of pulmonary gangrene, but which is due to decomposition of the sanguine fluid. Cold sweats, increasing dyspnoea, anxiety, and a frequent, thready pulse, precede death, the constant termination. Sometimes there is neither pain nor cough, but rapid increase of the breathlessness, and sudden death. The physical signs of the lung trouble are often wanting, though sometimes there may be dulness, mucous rhonchi, and bronchial respiration (HASPEL). Chest dulness on percussion in scurvy, may sometimes be due to sanguineous effusions into the thoracic muscles (HASPEL, BUZZARD).]

With our knowledge now of the phenomena of embolism, may not the suddenly fatal end of such cases be due to the morbid condition of the fibrine of the blood already referred to?

**Diagnosis.**—The scorbutic state of the skin is to be distinguished from *flea-bites*, *bruise*, *typhus fever*, and from *purpura hæmorrhagica*.

[A careful examination of the skin and gums, with a history of the case, will prevent its being mistaken for *chloro-anæmia*, though the color of the skin in the latter disorder, particularly when dirty, resembles that of a scurvy patient, but the blotches are absent. In *purpura* the blotches, though closely resembling those of scurvy, occur suddenly, in persons previously in apparent good health; while in scurvy they have been preceded by pallor and listlessness. In *purpura*, the dingy hue of the skin is wanting, also the articular effusions, and pains in the limbs.]

**Prognosis.**—In the present day, when the patient can command medical care and proper diet, scorbutus, though tedious, is seldom fatal. When these, however, have been wanting, the mortality has been terrible. Lord Anson, it should be remembered, in his voyage round the world, lost above 200 men, and at last could not muster more than six fore-mast men in a watch fit for duty. At the commencement of our last war with France, on the fleet returning from sea, it often happened so many men were landed ill of scurvy that even Haslar Hospital, large as it is, could not contain them, and many were lodged in the chapel, others in tents, while others died in the boats before reaching the shore.

**Cause and Conditions under which Scorbutus is Developed.**—In the Middle Ages scurvy prevailed to so great an extent that it was said to be epidemic among the inhabitants of the low countries of Holland, Friesland, Brabant, Pomerania, Lower Saxony, and, indeed, all countries from the 50° to the 60° of north latitude. This has been attributed to the absolute want of winter food for the cattle, so that it was necessary to kill them on the setting in of the frost, and either to salt or dry the flesh. Food was deficient and of improper quality, and hence the large stores of salt provisions found in the larder of the elder Spencer in the days of Edward II, even so late in the spring as the 3d of May. Six hundred bacons, eighty



carcasses of beef, and six hundred of sheep, was his abundant supply. In all these countries, however, in proportion as agriculture has advanced, and a succession of green crops has enabled the farmer to kill his best and fattest meats in winter, and in proportion as fresh vegetables have been introduced at our tables, together with a liberal use of wine and beer, so has this disease disappeared. The former universal prevalence of scurvy in the Navy, and its almost entire disappearance in the present day, necessarily has reference to a particular cause—the too exclusive use of salt provisions and absence of fresh vegetables. “In 1797 the victualling of the Navy was changed, greatly improved, and strictly regulated; and immediately consequent to the change the health of the seamen improved strikingly. Scurvy, typhoid fever, dysentery, and putrid ulcer, which up to the period of the change produced great havoc, became comparatively rare in occurrence and light in impression.” Since 1797 the improvements have consisted in giving cocoa instead of gruel for breakfast, issuing salt meats at a much earlier period after being cured, the supply of better articles, and in greater abundance by one-third, the substitution of tea in the afternoon instead of spirits; but, chief of all, the use of fresh vegetables as often as possible: and, with every improvement in these respects, there has been, as a general result, a further improvement in health, till these four forms of disease, at no distant date so destructive, are scarcely known except by name.

It is now, however, completely established that salted meats are not more productive of scurvy than fresh meats, so far as concerns a monotony of diet. The experience of the Russians in 1720 and 1736, of the French in 1750–60, and of our own regiments at the Cape in 1836, sufficiently establishes this point.

[There is no doubt, however, that in the great majority of scurvy outbreaks, salt meat has formed an important part of the food taken by the sufferers.

Liebig has shown that the process of salting meat deprives flesh of a large proportion of its most important constituents, so that the remainder is deficient in nutritive properties; and the altered and hardened character it acquires, renders even such nourishment as it contains difficult of assimilation.]

Besides the injurious effects of cold and moisture, as well as impure air, combined with the conditions already noticed as tending to favor the development of scurvy, it is now well known that exposure for a lengthened period to the pernicious influences of a malarious district greatly aids in developing scorbutus. By observations especially devoted to this subject, I have determined that, amongst our troops who had been in Bulgaria during the war with Russia in 1854, there were two and a half per cent. of admissions for scurvy amongst them more than among those who served in the Crimea only; and that the deaths among them reported from this disease were also greater by three per cent. than among those troops who served in the Crimea (*Glasgow Med.*

*Journal*, July, 1857; and *Trans. of the Royal Med.-Chir. Society*, vol. xl.)

Recent combined researches have shown that scorbutic diseases were developed amongst our troops in the Crimea under the following conditions: (1.) Deficiency of absolute nutriment; (2.) Improper adjudication of the *nutrient* and *respiratory* principles of the *diet*—its monotony; (3.) Bad quality of the diet, and improper cooking, or none at all; (4.) Exposure to cold, combined with imperfect clothing, and labor beyond the strength of the best fed men; (5.) The persistent pernicious influence of residence in a paludal district (Bulgaria). But these circumstances are only to be regarded as the *occasional antecedents*, any one of which, or all combined, can never, *per se*, originate the disease. LOOKING TO THE HISTORY OF SCURVY, IT WILL BE SEEN THAT THE INVARIABLE AND INDISPENSABLE ANTECEDENT OF THAT DISEASE HAS BEEN A DEFICIENCY OR ABSOLUTE WANT OF FRESH VEGETABLE FOOD. Privation of vegetable food is its one essential cause. There is no other invariable antecedent; and there are sufficient reasons why it may not always be followed by scurvy. It is this antecedent which is the *vera causa* of *scorbutus*; and the most successful methods of prevention and of cure are in accordance with the hypothesis which assumes it to be the cause (BUDD, CURRAN, LAYCOCK, PARKES). “The giving of vegetable food is its one essential counteractive” (SIMON).

[In 1846, the potato crop failed in Great Britain and Ireland; and in the following year there was much scurvy amongst all classes. There was an outbreak of the disorder in Scotland, especially amongst the artisans and the laborers on the railways, in 1846–47, described by Drs. Christison, Ritchie, and Lonsdale (*Ed. Monthly Journ.*, 1847), and the general fact with regard to the food of all was, that it failed in variety, and in the quantity of its animal constituents; none had tasted potatoes after the harvest of 1846—a period of seven or more months—nor fresh vegetables; but animal food, fresh and salted, was taken in large quantities, as well as pea-soup, suet puddings, bread, and oatmeal. At Workington, a seaport town of 7000 inhabitants, there was no case of scurvy, turnips having been used in large quantities. Dr. Curran (*Dublin Quar. Jour.*, 1847), describing the disorder as it appeared in Ireland, says: “In no single instance could I discover that green vegetables or potatoes had formed any part of the regular dietary;” grains, tea or coffee, flesh and fish, being the food. Dr. Shapter observes, that in Exeter “the only difference in the usual diet of the sufferers consisted in the absence of the potato,” and that many of them had abundance of the necessities of life, except fresh vegetables (*Med. Gaz.*, vol. iv).

In the Crimean war, the allied armies suffered severely from scurvy. In the British army it first appeared in Bulgaria, where the diet was poor, and the supply of vegetables scanty. When it arrived in the Crimea, there was an abundance of grapes, cabbages, &c., and though the ration was inferior, the disorder disappeared; but as winter set in, and vegetable food could no longer be got, it began again. When the supply of fresh vegetables and lime-juice became more constant, it gradually disappeared, and there were but few cases during the second winter.

The French army suffered still more, no less than 23,000 cases of scurvy being recorded (SCRIVE). Good but lean fresh meat was issued, at first



sequence. At one time, in the Army of the Potomac, when "symptoms of scurvy began to appear," and there was a general "low vitality of the men," Dr. Letterman found that he had rightly attributed it to "want of fresh vegetables;" for, "while large supplies of potatoes had been issued, the troops received in some cases a very small quantity, and in others none at all" (*loc. cit.*, p. 106). Again he says (pp. 109-10), "This favorable state of the health of the army, and the decrease in the severity of the cases of disease, are, in a great measure, to be attributed to the improvement in the diet of the men, by the issue of fresh bread and fresh vegetables, which has caused the disappearance of the symptoms of scurvy, that in January began to assume a serious aspect." The testimony of Dr. F. Hamilton, with respect to the Army of the West, is to the same effect (*loc. cit.*.)]

**Treatment.**—The early history of navigation, as it records the greatest ravages of scurvy, so does it also record the best antidote to the disease. Of four ships which sailed from England in the beginning of April, 1609, for the establishment of the East India Company, they were all so severely visited by scurvy as to have lost nearly one-fourth of their crews when they arrived at the Cape of Good Hope. The crew of the Commodore's ship was not attacked. This immunity arose from three tablespoonfuls of lemon-juice having been served daily to each of his men. But notwithstanding this evidence of the success of lemon-juice in preventing scurvy—evidence the most conclusive—this valuable remedy and preventive was altogether slighted for a hundred and fifty years afterwards (COPLAND). Lord Anson's people, in 1740, on reaching the Island of Tinian, were recovered principally by eating oranges; and that noble, brave, and experienced commander was so convinced of their usefulness that, before he left the island, he ordered one man from each mess to lay in a stock for future security. Sir Charles Wager's people, also, were terribly afflicted with scurvy in the Baltic. Sailing, however, in the Mediterranean, and having heard how effectual oranges and lemons were in the cure of this disease, he took on board at Leghorn a large quantity of them, ordered a chest each day to be brought on deck, and allowed the men, besides eating what they chose, to mix the juice with their beer, and to pelt each other with the rind, so that the deck was strewn with the fragrant liquor. By these means he brought his men home in good health.

In the year 1747 Dr. Lind made some comparative trials between this and some other modes of treatment (as vinegar, sulphuric acid, and tamarinds) on board the "Salisbury," at sea. As a general conclusion from his experiments, he affirms that orange- and lemon-juice, or more properly the citric acid obtained from all the species of the botanical genus *citrus*, or the natural order of fruits called *hesperidæ*, are greatly more efficient than any other remedy in the cure of scurvy.

Notwithstanding this strong opinion of Dr. Lind, the Navy continued to suffer severely from scurvy for half a century, till the Admiralty gave a general order for the supply of lemon-juice. This salutary measure was accomplished by a representation from the



disease, "so fatal when left to itself, is cured with the greatest facility. Symptoms apparently the most grave and serious vanish as if by magic, and without leaving behind them any serious injury to the constitution. The sanious discharge from scorbutic sores has been known to change color and to become healthy in a few hours after the commencement of treatment. In pure cases of scurvy the blood, and the blood only, is at fault." (PARKES, *l. c.*) "Lemon-juice," writes Dr. Watson, "is really a specific against scurvy, whether it be employed as a preventive or as a remedy.\* It supplies something to the blood which is essential to its healthy properties." The potato seems to be no less efficacious as a remedy and preventive (BUDD). The reader will find a most interesting account of the efficacy of potatoes and of onions in Dana's *Two Years before the Mast*—a book well worth reading. The antiscorbutic principle, whatever it may be, is in greatest amount in unripe fruits; it lessens gradually as they ripen; and if the juice be obtained, it disappears when fermentation occurs. When lime-juice becomes musty, a mucilaginous principle is developed at the expense of the citric and malic acids; and the *percentage* of citric acid gradually decreases (SIR WILLIAM BURNETT, PARKES). Good *lemon-juice* seems to be more effectual, however, than pure *citric acid*; probably from its containing *malic* and *tartaric acids*, besides *citric*, and from the *citric acid* being in the form most easily absorbed and decomposed by the digestive organs of man. The *Materia Medica* gives numerous analogous examples of the superior efficacy of a medicine in its *natural combinations* (PARKES). This is all we can yet say, however, regarding the actions of either of these means of cure, notwithstanding the researches of the chemists of the present day. Moreover, it is sufficient: and with such remedies at command, the prevalency of scurvy in *merchant vessels*, or in any navy, ought not to exist. "The one thing wanted in order that scurvy should be entirely banished from the *mercantile marine* is proper provision for the dietary of the crew,—such provision as is enforced in *emigrant ships*, where each person's weekly allowance must have in it 8 oz. of preserved potatoes, and

---

that the same cause will imperil the safety of our merchant ships. And there can be no doubt that many ships have actually foundered at sea because the crews were so prostrate from scurvy as to be unable to handle them when overtaken by severe weather. It has been the custom to inquire what proportion of the crews were disabled from scurvy. As might be supposed in the case of a disease resulting from a cause operating upon the entire crew, this proportion is often very large. Thus there were recently admitted on board the "Dreadnought" twelve cases of severe scurvy from one ship; two others were known to be seriously affected; and the entire complement of officers and men was only nineteen, leaving but five men in all able for duty to work the ship. The proportion of crew disabled has ranged from 20 per cent. to 70 per cent.; and it is certain that scurvy ships have rarely a hand to spare. Deprive such ships of a fourth, a half, or two-thirds of their force, and the peril of a ship, cargo, crew, and passengers, in stress of weather, is obvious; and it must not be forgotten that where scurvy has prostrated a large part of the crew, the vigor of the remainder is sure to be sapped, so that there may not remain a single sailor before the mast in a state of thorough efficiency (Simon, in *Sixth Report on Public Health*, p. 19).

\* [It is essential that the lemon-juice should be *pure*; and to insure this it should be analyzed, before being received either in the army or navy. Ten per cent. of brandy (s. gr. 930) or rum (s. gr. 899) ought to be added; and it should then be packed in jars, covered with a layer of oil, and sealed.—EDITOR.]



evidence not only that this is the case, but that their addition to the diet cures scurvy with great certainty. . . . Tartaric and especially citric acid, when combined with alkalies, have always been considered as antiscorbutic remedies, *par excellence*, and the evidence on this point seems very complete."\* The writer has found the bitartrate of potash very efficient in the treatment of scorbutic taint, and his experience is supported by the observations of others. Of the vegetable antiscorbutics, the *potato* enjoys, and probably deservedly, the highest reputation; sailors cut it in slices, and pack them in molasses; next to it are *onions*, sliced and eaten raw, which are greedily devoured by scurvy patients. *Cabbage*, in the form of *sourcrout*, *sorrel* (*rumex acetosella*), the *wild artichoke* (MADISON), the *maguey* or *American aloe* (*agave Americana*) (PERRIN), the *prickly pear* (*cactus apuntia*), the *dandelion* (*leontodon taraxacum*)—much used and prized by the French army in the Crimean war—*lamb's-quarter* (*chenopodium album*), *green corn* (SOUTHWORTH, HAMILTON), the *yam*, *apples*—not *cider*—have all been found excellent antiscorbutics. The expressed juice of the *sorghum*, before boiling, was much used in the Southern army, and it is stated, with excellent results. It is unquestionable, that uncooked fruits and vegetables are more efficient than cooked, in the prevention and cure of scurvy. Dried or desiccated vegetables are greatly inferior in antiscorbutic properties to fresh (MARRAIN); and there is always great difficulty in getting soldiers to use them, they having a distaste for them, owing, in a measure, to ignorance of the proper way of preparing them for the table. The writer has seen much benefit from the use of the water in which the desiccated potato had been soaked and boiled, regularly administered to scorbutic men. Fresh or even raw meat, and new milk, are important articles of diet in scurvy, which is ascribed to the lactic acid they contain, an attribute not proved. Heat and light are powerful auxiliaries in the treatment of scurvy; and exposure to the sun's rays, the head being covered, has a salutary effect. In the sclerotic condition of the subcutaneous tissues and muscles, frictions with camphorated lotions, shampooing, alkaline and sulphur baths, have been used with good results. The spongy gums and ulcerated spots should be sponged with solutions of tannin, chloride of iron, or diluted nitrate of silver (BUZZARD). Fetor is removed or abated by a wash containing the permanganate of potash. The muriated tincture of iron, with arsenic and quinine, and phosphoric acid, should always be liberally given.

Dr. Parkes advises that the following measures be adopted in time of war, or in prolonged sojourn on board ship, or at stations where fresh vegetables are scarce:

1. The supply of fresh vegetables by all means in our power. Even unripe fruits are better than none, and we must risk a little diarrhoea for the sake of their antiscorbutic properties. In time of war *every* vegetable should be used which it is safe to use, and when made into soups all are tolerably pleasant to eat.

2. The supply of dried vegetables, especially potatoes, cabbage, and cauliflowers; turnips, parsnips, &c., are perhaps less useful; dried peas and beans are useless. As a matter of precaution these dried vegetables should be issued early in the campaign, but should never supersede the fresh vegetables.

3. Good lemon-juice should be issued daily (1 oz.), and it should be seen that the men take it.

---

\* [Brit. and For. Med.-Chir. Review, 1848. Practical Hygiene, 1866.]





found on the serous membranes, as in the *arachnoid*, the *pleuræ*, the *pericardium*, the *peritoneum*; and occasionally the blood lies coagulated in the cavities of these membranes. It has also been found in the bladder and in the calyces and pelvis of the kidney (CRAIGIE).

Instances occur in which blood oozes, or is discharged more or less copiously, from the mucous membranes without affection of the skin; and to this class of cases, as a form of disease now under consideration, are to be referred those cases of profuse or fatal hemorrhage from slight causes, recorded under the name of "*hemorrhæa*," which have been ascribed to a *diathesis* termed the *hemorrhagic*, and which is supposed to be hereditary (*Edin. Med. and Surg. Journal*, vol. xxv). When the disease is protracted the patient becomes of a sallow complexion, waxy-colored, and dingy; anasarcaous swellings, gangrenous and bed sores appear on the feet and legs, general anasarca prevails, and he dies exhausted. Dr. Craigie considers that the appearance of purple spots in such cases is connected with more or less disease of the heart, especially with hypertrophy or dilatation of the ventricles. The following observation by Dr. Watson puts the pathology of purpura in an interesting and practical light:

"I have adverted to one peculiar source of danger in purpura, the hazard that blood may be effused in some vital organ, where even a slight amount of hemorrhage suffices to extinguish life. Dr. Bateman states that he had seen three instances in which persons were carried off, while affected with purpura, by hemorrhage into the lungs. During the course of one week, in the year 1825, I was present at two inspections in the dead-house of St. Bartholomew's Hospital, illustrative of the same point in respect to another vital organ, and involving a question in forensic medicine. The subjects of examination were both of them women of middle age, who had been brought into the hospital covered with purple spots and bruise-like discoloration, and suffering hemorrhage from the mucous membranes. Each of these women declared that the apparent bruises were marks of beatings received from her husband. One of them became suddenly hemiplegic a little while before she died. Of the manner of dissolution in the other case I am not sure. In both instances a considerable quantity of blood was spread over the surface of the brain between its membranes; and in one of them blood had been shed also into the cerebral substance, which it had extensively lacerated.

"It may be worth mentioning that in one of these corpses there were indications either of unusually rapid putrefaction after death, or (what I think more probable) of some degree of decomposition even before life was extinct. This woman died in the evening, and the body was examined next day, twelve or fourteen hours afterwards. A quantity of fetid gas escaped from the cavity of the abdomen as soon as it was opened, and small bubbles of air were seen to ooze from the areolar tissue of various parts of the body. Even when incisions were made into the *liver*, air frothed up, as it might do under ordinary circumstances from a section of the lungs" (*Lectures*, vol. ii, p. 866).

**Symptoms.**—Various symptoms, denoting general disorder of the constitution, precede the appearance of the petechiæ, generally by some weeks, such as languor, which is oppressive, weariness, faint-



*trembling*, with an extraordinary *thrill* and *hardness*, “yielding only to the withering influence of approaching death.” Bleeding from the intestines and urinary system quickly supervened. These phenomena continued to spread till the whole mucous system of the patient became the seat of copious hemorrhage. A peculiar exanthematous eruption appeared upon the skin, lasting for several days (five), and never presenting any signs of extravasation. Death followed in about four weeks. The pulse did not exceed its natural frequency (70), and there was no disturbance of the nervous system, such as headache, delirium, or want of sleep; and in the beginning of the disease the strength was not remarkably impaired; but subsequently yielded to loss of blood. The disease seemed almost exclusively confined to the arterial and capillary systems of vessels.

**Treatment.**—To treat this disease with success, it is necessary to ascertain the circumstances under which it becomes developed in each particular case. *Quinine* or *bark*, stimulants, *mineral acids*, *nutritious food*, and *wine*, was the treatment adopted by Cullen, Duncan, and Willan. Subsequently this plan of treatment was questioned by Dr. Parry, of Bath, who found that in his cases a full bleeding from the arm was a much more speedy mode of curing the disease. Dr. Harty, of Dublin, confirmed this principle of treatment, and was successful with liberal doses of purgatives, so as to clear out the intestinal canal completely. *Oil of turpentine*, administered in moderate and repeated doses, has also been recommended. Dr. Hardy, of Dublin, recommends the tincture of *larch bark*. He has long used it as a styptic and carminative tonic; and it is “one of the most elegant forms of prescribing a terebinthinate” (MOORE). *Fifteen-drop doses* of the tincture may be administered every hour, or *eight or ten drops three times a day*, afterwards increasing the dose according to the age of the patient and the necessities of the case.

The treatment of purpura may be comprised in the following measures: The bowels ought invariably, and without exception, to be first thoroughly and effectually evacuated by means of senna, aloetics, or calomel and jalap. If several effectual doses of either or all of these medicines be not followed by less heat of skin, diminution of the frequency of the pulse, abatement of the internal pains, and a cleaner state of the tongue; if the spots continue to increase in number and size, and the hemorrhagic oozings do not cease,—blood, according to age, strength, and other circumstances, may be drawn from the arm, while the patient should abstain from animal food in every form, and should subsist on boiled rice with whey, or the light subacid fruits, as grapes, oranges, strawberries, gooseberries, baked apples, or the like. His drink may consist of tamarind-water, or water acidulated with sulphuric acid. Under this plan most cases of the disease will be speedily and readily brought to a favorable termination. If symptoms of local uneasiness continue after the urgent phenomena have disappeared, leeches should be applied in the neighborhood of the part; and it will be proper to continue the periodical and regular evacuation of the alimentary canal. When the spots have disappeared and the hemor-



sometimes rose to the upper part of the thigh. In some patients the gangrened part came away of its own accord; in others it became necessary to assist nature by amputation. In some instances death succeeded to amputation, the disease having continued to extend to the trunk of the body. It is particularly mentioned in a report on the subject to the Royal Academy of Sciences at Paris, that the rye of Sologne, in the year 1709, contained fully *one-fourth* part of the cockspur. The disease generally began in one or both feet, with pain, redness, and a sensation of heat as burning as the fire. At the end of some days these symptoms ceased as quickly as they had come on, when the sensation of extreme heat was changed to cold. The part affected became black, like a piece of charcoal, and as dry as if it had been passed through the fire. A line of separation tended to form between the dead and the living parts, like that which appears in the separation of a slough produced by the application of the cautery.

The disease prevailed in Switzerland in 1709 and 1716, and is described by Langius, of Lucerne. M. Garroud, a physician of Dauphiny, where the disease prevailed in 1709, makes some very important observations on the different symptoms apt to predominate in different individuals. Some patients suffered very violent pain, with an insufferable sensation of heat, although the part felt cold to the touch. In other patients redness, with much swelling, supervened, attended by fever and delirium. The separation of the dead parts from the living took place with excruciating pain. The gangrene was not in every instance dry. Animals were found to die of the specific gangrene when forced to swallow the diseased *rye*, for they refused to eat food containing it.

The experiments and observations of Tessier show that a given quantity is required to produce the specific effects, and that the action of the poison is cumulative.

The history of some cases of mortification of the limbs related by Dr. Charlton Woolaston, in the *Philosophical Transactions* for 1762, shows that it may occur in this country from eating *wheat* diseased similarly to the *rye*; and Sir William Wilde, of Dublin, has recorded its occasional occurrence in Ireland.

**Symptoms.**—The gangrenous form of ergotism is ushered in by excessive lassitude, more or less protracted, and accompanied with fever; the extremities become painful, cold, and rigid, benumbed, and almost insensible, and are with difficulty capable of movement. Severe internal pains of the limbs prevail (acrodynia), greatly aggravated by heat. It extends by degrees from the toes to the legs and thighs, and from the fingers to the arms and shoulders, when sphacelus supervenes. With the exception of slight febrile heat, the constitutional disturbance appears to be slight, and in this respect resembles scurvy. Ricker has recently described the early symptoms of a case of poisoning by bread containing *ergot*. A family of six persons partook of the bread, and all suffered from the same symptoms—namely, dryness of the throat, epigastric oppression, nauseous taste, mucous and biliary vomiting, vertigo, stupor, and diarrhœa (*New Syden. Society Year-Book*, 1861). Seeing that symp-

toms vary greatly in severity, probably in proportion to the amount of diseased food taken, and the poisonous nature of the particular fungus which affects it, we may have the expression of some phenomena more fully than others—for example, *acrodynia*. Under this name Chomel described a painful affection of the wrists and ankles which prevailed in Paris in 1827, 1828, and 1829. It was then so prevalent among the soldiers in the barracks of Lourcine, that 560 men were affected out of 700; and in that of Comtille, 200 men out of 500. Since 1828 no case has been recorded in the barracks or military hospitals (PARKES). In 1859 M. Barudel observed three cases in the military prison at Lyons; and in spite of negative results of the examination of the quality of the food, Dr. Parkes is inclined to believe that the cause lay there, and probably in ergotism of the flour (*Army Medical Department—Sanitary Report for 1860*, p. 358).

The general train of symptoms produced by the use of diseased grain assumes two forms—namely, the spasmodic or the gangrenous. The spasmodic form commences with a sense of tingling or itching in the feet, followed by cardialgia and similar tingling sensations in the hands and head. Violent contractions of the hands and feet follow, which seem to affect each particular joint, and the pain is said to resemble that of a dislocation. The sensations are also sometimes described as of a bruising kind; and the body is bathed in copious sweats. The symptoms intermit during intervals of two or three days of a remission at one time. Drowsiness, giddiness, indistinctness of vision, and an irregular gait are constant phenomena. Coma and epileptic convulsions are apt to supervene, which generally indicate a fatal result. An enormous appetite accompanies this train of evils. Spots like those of purpura appear on the face, and the disease rarely abates before the third week.

**Treatment.**—Considerable differences of opinion prevail regarding the treatment of this *dietic* disease. The cause, in the first instance, must be ascertained and removed. To obviate the effects it has already produced, the constitutional treatment must be directed to improve the state of the blood. Tonics and stimulants are to be administered after a free employment of evacuant remedies to clear out the alimentary canal completely. The chlorates of potash and of soda, with antispasmodics, tonics, and narcotics, are especially indicated. *Camphor*, *musk*, *ammonia*, *capsicum*, may be particularly mentioned; and the strength of the patient is to be supported by light, nourishing, and wholesome food.

#### DELIRIUM TREMENS.\*

LATIN Eq., *Delirium alcoholicum*; FRENCH Eq., ———; GERMAN Eq., *Delirium tremens*—Syn., *Säuferwahn sinn*; ITALIAN Eq., *Delirium tremens*.

**Definition.**—*A train of morbid phenomena, produced by the slow and cumulative action of alcohol, in the various forms in which it is used as*

---

\* [The term *Delirium Tremens* was first proposed by Dr. Sutton (*Treat on Delirium Tremens*, London, 1813).—EDITOR.]

a drink. *Delirium* is one of the most prominent features of the morbid state, which is otherwise characterized by hallucinations, dread, tremors of the tendons and muscles of the hands and limbs, watchfulness, absence of sleep, great frequency of pulse. A thick, creamy fur loads the tongue, and a cool, humid, or perspiring surface prevails; while the patient gives forth a peculiar odor, of a saccharo-alcoholic description, more or less strong.

**Pathology.**—This disease has only been recognized and described since the beginning of this century. The *Experimental Inquiry* of Dr. John Percy, in 1839, illustrating the physiological action of alcohol; an inquiry into the *Physiology of Temperance*, by Dr. Carpenter; the recent *Pathological Observations on the Bodies of Known Drunkards*, by Drs. Roesch and Francis Ogston (1855); and, lastly, a most able and interesting review on the "Treatment of Delirium Tremens," in the *Brit. and For. Med.-Chir. Review* for October, 1859, are contributions which have placed on a more sure foundation our previous theoretical information regarding morbid states which follow the persistent use of alcohol.

The term *alcoholism*\* is used to denote various symptoms of disease attending morbid processes of various kinds which are capable of being traced to the use of stimulants containing alcohol. The immediate effects of *intemperance* in the use of alcoholic fluids, the nature of *delirium tremens*, and of *spontaneous combustion*, may be embraced under the general designation of *alcoholism*. This term is used in the sense analogous to that in which we use the terms *mercurialism*, *ergotism*, *narcotism*, and the like;—the agents inducing these specific states acting after the manner of a cumulative poison. The progress of modern science has distinctly demonstrated the poisonous action of alcohol; and an account of the nature of *delirium tremens*, as well as the grounds on which its treatment must be based, are now alike founded on this knowledge. Tiedemann and Gmelin in 1820, and Magendie in 1823, detected alcohol by its odor in the blood. The fluid found in the ventricles of the brain had also been observed to have the smell, the taste, and the inflammability of gin (SIR A. CARLISLE). In 1828 it was theoretically advanced by Leovèille that *delirium tremens* consisted in an exalted state of the vital powers of the brain, excited by molecules saturated with alcohol absorbed from the surface of the stomach and bowels, and carried into the current of the circulation. Now it is a matter of fact, determined by direct experiment, as well as by casual observation, that alcohol is absorbed directly into the circulation, and is capable of acting as a direct poison upon the nervous tissue through which the infected blood circulates. Alcohol has been found in the blood, in the urine, in the bile, in the fluid of the serous membranes, in the brain-matter, and in the liver (PERCY, OGSTON). Its odors can be easily detected in the breath, and the habitual immoderate drinker exhales a distinct alcoholic and saccha-

---

\* [The term *Alcoholism* was first used by Requin (*Eléments de Pathologie Médicale*, t. iii, January, 1852). In the same year Professor Magnus Huss, of Stockholm, published his work, *Die Chronische Alkohols Krankheit*].



rine odor more or less strong. His clothes at last acquire a spirituous aroma, every part of his body being long thoroughly imbued with alcohol (CRAIGIE). This odor is generally so well expressed in cases of *delirium tremens* that a place has been given to a statement of the facts amongst the characters embodied in the definition. Dr. Percy's experiments directly support these statements, and prove at the same time the great rapidity with which alcohol passes into the current of the circulation. He injected strong alcohol into the stomachs of dogs; and within two minutes after completing the injection, their respiratory and cardiac movements ceased; the stomach was found nearly empty after death, whilst the blood was highly charged with alcohol.

I once had an opportunity of examining the body of a person who for many years had been in the habit of drinking daily a large quantity of brandy. He died of *typhoid asthenia*, with characteristic degeneration of nearly every important organ of the body and of the bloodvessels. The fluid collected from the cavities of the brain, consisting of serum and some blood, contained 2.6 per cent. by volume, and 2.1 per cent. by weight, of alcohol. This quantitative analysis was made for me by my friend Dr. F. S. B. F. de Chaumont, with Giessler's vaporimeter.

The pernicious effects of the continuous use of alcoholic stimuli on the organs and tissues of the body have been deduced from a careful study of the morbid appearances, of a *chronic kind*, met with in the bodies of individuals known to have lived intemperate lives, and who *had perished suddenly* from the effects of accident, suicide, or homicide, and while apparently in ordinary health and activity. The extent of the chronic changes in the various organs of these individuals is found to have been far in excess of what could have been reasonably looked for in a like number of persons of the same age, and of temperate habits, suddenly cut off while apparently in average health and vigor. The *cumulative* effects of long-continued intemperance have been clearly proved by Dr. Ogston's observations; and the results of his post-mortem inspections, on the whole, support the conclusions which have been arrived at on *theoretical grounds* as to the injurious effects of alcohol in excess. The following statements contain a summary of these results: (1.) *The nervous centres* present the greatest amount of morbid change, the morbid appearances within the head extending over 92 per cent. of those examined. By this observation the theoretical remarks of Leovèille, Craigie, and Carpenter are clearly established. (2.) *The changes in respiratory organs* succeed in frequency those of the nervous centres, yielding a percentage of 63.24 of those examined. (3.) *Morbid changes in the liver* are next in order of frequency, and are due to enlargement, granular degeneration, the nutmeg-like congestion, and, lastly, the fatty state. (4.) Next to changes in the liver come those in *the heart and large arteries*. (5.) Next are those of *the kidneys*. (6.) Least frequent of all are morbid changes in *the alimentary canal*.

Two orders of changes may be observed to result from intemperance in the use of alcoholic fluids—namely, one set of long duration, or which at least must have taken some considerable time

before they could be completed; another set of shorter duration, and which probably are more closely connected with the immediate symptoms which precede the fatal event.

The abnormal changes in the cranium, the substance of the brain, its convolutions and cerebral ventricles, all indicate the prolonged action of a morbid poison. The prolonged action of the alcoholic poison on the cranial contents is to produce induration of the cerebral and cerebellar substance in by far the largest number of cases, coincident with an increased amount of subarachnoid serum; while the steatomatous degeneration of the small arteries leads to *atrophy* of the convolutions and *œdema* of the brain.

When spirituous liquors are introduced into the stomach they tend to coagulate, in the first instance, all albuminous articles of food or fluid with which they come in contact: as an irritant, they stimulate the glandular secretions from the mucous membrane, and ultimately lead to permanent congestion of the vessels, to spurious melanotic deposit in the mucous tissue, and to thickening of the gastric substance. By the veins and absorbents of the stomach the alcohol mixes with the blood, and immediately acts as a stimulant to all the viscera with which it is brought in contact. The functions of the brain are at once stimulated, and ideas follow in more rapid succession; the liver is excited to secrete an excess of sugar, by the immediate action of the stimulant on its tissue (Drs. HARLEY and BERNARD). The flow of urine is excited in a similar manner.

In these effects it is impossible not to recognize the operation of an agent most pernicious in its ultimate results, when indulged in habitual excess; but most valuable as a remedial agent, when its action is understood and appreciated (consult Dr. Anstie's valuable work on *Stimulants and Narcotics; their Mutual Relations*). The mere coagulation of the albuminous articles of food and fluid is very different from that effected by the gastric fluids, and tends to render the articles more difficult of solution by the gastric juice.

In these facts it is impossible not to recognize that alcohol being absorbed, a double series of morbid results ensue. On the one hand, a train of phenomena are induced partly of a *chemical* nature and partly *physiological* or *vital*. The general nutrition of the body suffers; and a bad state of health is at last induced, of a peculiar kind, sometimes described as the "*drunkard's cachexia* or *dyscrasia*." This state of the system is characterized by positive irritation, which very soon succeeds to the intemperate use of alcohol, and which is manifested in a variety of ways; sometimes by an unnaturally voracious appetite; but those who over-indulge in the use of such stimuli subsequently suffer a total disrelish for food; they become unable to eat, and dyspeptic symptoms of various kinds betray the irritable state of the alimentary canal, such as stomach-ache, the frequent generation of gases, water-brash, heartburn, squeamishness, vomiting, and palpitations of the heart. A constipated condition of the intestines, attended with deficiency in the power to expel their contents, is very soon established, and sometimes ascribed to the deficient secretion of bile, which is known not to be secreted in due



eliminated very slowly as alcohol by the lungs, by the liver, and by the kidneys; but appears to tarry in largest amount in the liver and in the brain (PARKES). Another portion is decomposed. Its hydrogen enters into combination with oxygen to form water, which, with acetic acid, having been produced, carbonic acid and water are formed. Oxygen is thus diverted from its proper function, the exhalation of carbonic acid at the lungs is diminished both absolutely and relatively, and less urea is excreted by the kidneys than is consistent with health; but the pulmonary aqueous vapor is not lessened (BÖCKER and HAMMOND, quoted by PARKES). The water of the urine is diminished, the chlorine is greatly lessened, as well as the acids and bases. All the evidence, therefore, points to the effect of alcohol as causing the retention of substances which ought to be eliminated; and this retention of the effete matter is still more intensified by the stimulant action of alcohol increasing for a limited time the frequency of functional acts, followed as it is by a corresponding depression. In this way impaired health is soon brought about, tending to wasting of the tissues generally; and so long as any alcohol remains in the blood *as alcohol*, a certain toxic or poisonous effect continues to be produced upon the nervous system through which the poisoned blood circulates. If a constant supply of the alcohol is kept up, the phenomena of *alcoholism* becomes chronic or persistent; and acute paroxysms, generally in the form of *delirium tremens*, supervene, which is at once the most common and the most prominent evidence of *alcoholism*. In other instances the degeneration of several vital organs generally may become so excessive that death follows by *asthenia*, or with typhoid phenomena ending in coma. [See *Chronic Alcoholism*, by the Editor, vol. ii.]

When mixed with blood out of the body, spirituous liquors cause more or less coagulation, according to their strength and concentration; and when applied to the bloodvessels in the transparent parts of animals, they can be seen to produce the same effects. The congestion that constantly exists in the mucous membranes of the lungs and stomach is evidence of the retarded motion of the blood. The fact that hemorrhoidal swellings are always aggravated by the use of alcoholic fluids is the result of retarded motion of the blood in the hemorrhoidal vessels. When death occurs from poisonous doses, either in animals or in man, although the dose is at first followed by increased frequency of the pulse, yet in a short time the pulse becomes rapid and small; while the extremities become cold, and the power of generating heat is suspended in proportion as the blood progresses slowly and more slowly through the pulmonary capillaries. These effects upon the lungs must be regarded as of a toxic kind; and this specific toxic action is not less obvious on the brain. Its nerve-substance becomes poisonously affected—a condition which seems to constitute one of the most necessary antecedents in the causation of *delirium tremens*.

The effects produced on the medulla oblongata tend to sustain this toxic effect upon the lungs. The brain and the lungs in this respect act and react on each other. Death may ensue in various



his affairs, and is either restless or watchful. These symptoms last from twenty-four to forty-eight hours.

The second stage commences by a hurried and anxious manner, by great excitability of temper, by a small, accelerated pulse; some heat, perhaps, of the surface of the trunk, but accompanied with the coldness and clamminess of the extremities. The tongue is sometimes clean, but often brown and dry, and the patient delirious, suffering from various mental illusions and alienations. In general the delirium is melancholy, and has reference to his usual occupation and habits, or to some difficulty in his domestic affairs. He sometimes sees flames or hears voices talking to him, or as soon as he shuts his eyes he sees people passing under the bed-clothes. In short, he sees objects and sights in situations in which they are not, and which have no real existence; or betrays the most dreadful alarm at hideous objects which he imagines are threatening him with immediate destruction. Restless and sleepless, he moves his trembling hands horizontally over the bed-clothes, as if seeking for something. In general he is harmless and easily controlled; but in some instances he is violent, mischievous, and requires to be restrained. This stage generally lasts from three to four days to a week, when the third stage commences by the patient falling into a sound sleep and gradually recovering, or a fatal collapse comes on, which finally and shortly closes the scene. Without reference to stages of the disease, the following is a general description of its symptoms.

There is always more or less derangement in several other functions besides the brain. The patient is generally void of all appetite; or he may be squeamish, and vomit at intervals. Sometimes he is thirsty, and calls loudly for liquor of various kinds; but often he is indifferent to the sensation of thirst. In several instances great aversion, and even dread, of all food and drink has been evinced; and it has been impossible to persuade the patient to partake of either. The tongue is at first covered with moist white, gray, or slate-colored fur, and when protruded it is tremulous. The bowels are constipated, and less sensible than in the state of health to the action of medicine. When they are acted upon by remedies, the discharges are very dark-colored, the first generally consistent, the latter liquid, dark, and offensive. There are generally fulness and distension, and not unfrequently tenderness and pain, in the epigastric, umbilical, and right hypochondriac regions; and sometimes the two hypochondriac regions give the patient the sensation as if they were drawn tightly together. The skin is bathed about the head and neck with a clammy, unctuous, cold moisture; but elsewhere, and especially at the feet, it is cold, dry, and imper-spirable.

The pulse varies from 96 to 110 or 120, sometimes 130; and though sometimes small and oppressed, is often full, voluminous, and throbbing. The carotid and temporal arteries beat most violently; those of the wrist less forcibly; and the anterior and posterior tibial arteries pulsate feebly enough. The action of the heart



The late Sir Alexander Tulloch, in his report for 1853, gives the following percentages of mortality among them:

Great Britain, infantry,	. . . . .	17.6
“ cavalry,	. . . . .	18.8
Bermuda,	. . . . .	15.0
Canada,	. . . . .	7.94
Gibraltar,	. . . . .	18.6
Malta,	. . . . .	8.8
Nova Scotia,	. . . . .	9.1

A return of admissions and deaths from *delirium tremens* and *ebrietas* in the General Hospital in Calcutta, from 1848 to 1852, and another of admissions and deaths from the same causes in the Medical College Hospital, during 1851, 1852, and 1853, give some important results, as follow:

That *delirium tremens* occurs in women and men in the proportion of one to twenty-five; but that this difference is due to the difference of habits rather than of sex.

That in regard to age the ratio is as follows:

	Cases.	Deaths.	Per cent. of Deaths.
Ages from 20 to 25,	84	4	9.1
“ 25 to 30,	66	16	24.2
“ 30 to 35,	48	11	22.9
“ 35 to 40,	76	7	9.2
“ 40 to 45,	62	6	9.6
“ 45 to 50,	28	4	17.8
“ 50 to 60,	7	—	—
“ 60 to 65,	5	1	—

The greatest mortality is between the ages of twenty-five and forty, which is confirmed by the analysis of another series of sixty-four fatal cases. The percentage shows that there is no uniformity in the proportion of deaths to the number of cases.

There is no evidence to show that the season of the year exerts a definite influence on the occurrence of the disease, whereas the mortality very palpably varies with the temperature—it being more than double in the eight hot months as compared with the four cold months.

The apparent cause of death was as follows: Thirty-three by exhaustion (often with coma); eighteen by coma; eleven by fits (probably apoplectic, called sometimes epileptic); one died on the night-stool; one was found dead in bed.

Convulsions occurred in at least twenty of the above cases. One distinct case of paroxysmal *opisthotonos* occurred in a musician, who during the intervals was able to sit up and whistle tunes.

**Treatment.**—From the nature of the disease as now described, as well as from the dire results of experience, it is now clearly established that the indications for treatment are,—(1.) The elimination of the poison; (2.) The sustenance of the patient during this period. The two most fatal errors which can be committed in the treatment of *delirium tremens* are either to *bleed* the patient or to give him *opiates*. The greatest number of cases of those treated by opiates





tion ; and a laxative, or even a purgative, should be alternated with opium, followed by a bitter tonic, which always operates favorably in lingering cases of nervous and dyspeptic exhaustion. Narcotics are thus only safe in *delirium tremens* when they are given with the object of aiding and seconding the natural cure of the disease, employed in moderate doses, and given only at the later stages. The heroic use of them, as heretofore too often advocated even by the most eminent physicians, is now recognized as a treatment which merely substituted narcotic poisoning for *alcoholism* or *delirium tremens*.

### LEAD PALSY.

LATIN Eq., *Paralysis ex plumbo* ; FRENCH Eq., *Paralysie saturnine* ; GERMAN Eq., *Beilähmung* ; ITALIAN Eq., *Paralisi litargirosa*.

**Definition.**—*A series of morbid phenomena induced by the absorption of the salts of lead contained in solution in drinking-waters, or in various foods and drinks, or conveyed into the system through the integuments of those who are in the habit of handling the soluble salts of lead ; or through the pulmonary mucous membrane of those exposed to the influence of vapors containing lead.*

**Pathology.**—The theory of this disease is, that the lead being absorbed, produces a peculiarly painful affection of the alimentary canal, termed *lead colic*, or *painters' colic* (eq., *colum ex plumbo*). It may also affect the muscles of the extremities, producing palsy ; and finally it produces ulceration of the gums and alveolar processes, accompanied by a peculiar blue line, which was first pointed out by Dr. Burton, of St. Thomas's Hospital. This blue line is seen along the free margin of the gums, but is absent where a tooth or stump is wanting. To this the name of "blue gum" has been given. A stain, also from lead, sometimes affects the conjunctiva.

The fact of the lead being absorbed and mingled with the blood is demonstrated by the circumstance that lead has been obtained from the coats of the stomach of a dog poisoned by lead, even as late as a month after poisoning. Again, MM. Duvergie and Guibourt have detected lead in the brain of the human subject, and Dr. Budd has detected it, not only in the human brain, but also in the muscles. Many pathologists are inclined to believe that the blue line observed in the gums of persons poisoned by lead is owing to the presence of lead in some peculiar state of combination, as with some of the constituents of the tartar of the teeth (TOMES) ; and from the observations of the late Dr. George Wilson, it appears that there are various tissues of the body for which lead has an affinity, and that it is more apt to be found in some organs than in others. The stomach and cæcum of a pony that died a fortnight after being removed from the sources whence lead was received into its body through the ingesta, having been carefully analyzed by this able chemist, it was found that, while the contents of these viscera did not contain the metal, the substance of their tissues



sulci between them wide, and sometimes patches of white softening are to be seen in the hemispheres; and this seems to be more particularly the case in those who have had paroxysms of an epileptic nature, and in whose brains lead has been detected (TODD).

The introduction of lead into the system has taken place in a great variety of ways. In France the pernicious effects were wont to occur from putting a lump of litharge into the *vin gâtée*, to cover its acidity and render it salable; and from this having been practised to a great extent by the Pictones, or the inhabitants of Poitou, the disease has been named *Colica Pictonum*. In the cider counties of Great Britain this disease formerly existed to a great extent, and has been termed Devonshire colic, or *Colica Damnoniensis*. The impregnation of cider with lead in this country was generally the effect of accident, and arose from the troughs in which the apples were crushed having the different pieces of stone of which they are composed clamped together with iron, and fixed by melted lead. In some districts it was the practice to line the entire press with lead, or to tip them with that metal. It was a custom, also, almost universal, to make the upper part of the boiling vessel of lead; while some growers, in managing weak ciders, put a leaden weight in the cask to sweeten the liquor. From these and perhaps other causes, Sir George Baker found the cider he examined to contain four and a half grains of lead in eighteen bottles, or a quarter of a grain in each bottle. In the West Indies lead poisoning appears to have been produced by using leaden worms to the stills, by which the rum became impregnated with this metal. There are many other minor sources of poisoning by lead; as keeping pickles or preserves in glazed earthen vessels, and coloring confections with preparations of lead. The still and other machinery used in the distillation of fermented liquors being now constructed of metals so combined as not to be acted upon by acid fruits or sugar, diseases from the action of lead are no longer so common as they were wont to be, but are confined principally to laborers in the lead manufactories and to painters. The use of paint where lead exists is the most common source of its absorption in this country; and hence house painters are those most frequently affected. The paint called "flatting" (or that which is mixed with a large amount of turpentine, so as to give a *flat, dead, or non-glistening* surface) is the most injurious to the workman. The turpentine, readily passing off by evaporation, carries with it a small supply of lead, which is constantly and gradually inhaled, or it is left on the skin to be absorbed, or mixing with saliva, it gets into the stomach. By one or all of these ways the system becomes affected, first through the circulation of the blood, and subsequently by the constituent tissues of the organs combining, in some form or other, with lead, which is thus deposited in them.

All ages, both sexes, and all classes are liable to the poisonous action of this metal; but the workers in lead have been at all times the greatest sufferers. Women in this country often suffer from lead colic, but it is rare to find them paralytic; men suffer both from the colic and the characteristic palsy.



of the thumb are also in a similarly wasted state. In general both arms are palsied, but not equally so, one being slightly more affected than the other. Supposing both sets of muscles to be equally palsied, the patient usually recovers the use of the *flexors* before that of the *extensors*, so that he can carry a weight hanging in his hand before he can shave himself. This restoration of the lost power is usually accompanied by more or less pain. The duration of the palsy under any treatment is always long, and often lasts many months, and in some cases years. Both colic and palsy may occur an indefinite number of times. When epilepsy is produced, the fit does not differ from epilepsy due to other causes.

**Diagnosis.**—The colic of lead poisoning can only be distinguished from ordinary colic by the history of the case, and by the blue line on the dental edge of the gums, but which is present only where the teeth or their stumps are in the alveoli, and ceases where a tooth is completely wanting.

The palsy is to be distinguished from cerebral paraplegia by the history of the case, by the integrity of the intellect, and by the blue line on the gum. A most important means of diagnosis in paralytic affections is the electric current properly administered. The excitability of the muscles is always much diminished in paralysis from lead, as Dr. Althaus clearly shows, and often it is entirely lost. Such is the case, not only when the muscles are atrophied, but when the bulk of the muscles is only slightly diminished; and even after the voluntary movements have regained their former power, the excitability of the muscles to the electric current still remains impaired. The relation of the muscles to the stimulus of Faradization helps in doubtful cases to establish the diagnosis, as the excitability of the muscles is always either lost or diminished in lead palsy, whilst it is normal in spontaneous paralysis. *Therefore, when the muscles of a paralytic limb move well under the influence of the electric current, we may fairly conclude that there is no lead in the system* (Althaus *On Paralysis, Neuralgia, &c.*, p. 72).

**Prognosis.**—The termination of lead colic, except where the dose has been in such excess as to produce death in a few hours, is always favorable; and those cases which prove fatal are generally such as have been exposed to the cumulative influence of lead for a long time, and who have been intemperate.

The palsy does not appear greatly to affect the health of the patient; but in some cases it has hitherto not been cured or relieved. In general, however, the patient recovers, although perhaps not completely. Drs. Garrod and J. W. Begbie have satisfactorily demonstrated that lead poisoning of the system exerts a remarkable influence as a predisposing cause of gout; and my friend, Dr. W. England, of Winchester, as the result of his experience, spontaneously volunteered to me a similar remark. Inveterate forms of dyspepsia may be traced in many cases to the influence of salts of lead in the drinking-water, its pernicious influence expressing itself differently in different constitutions, although never amounting either to colic or to paralysis.



retained among them as an *insoluble* compound. The *iodide of potassium*, after its absorption into the blood, combines with the lead, and forms with it a new and soluble salt. The poison is thus liberated from its union with the injured part, dissolved out from the damaged fibre, and once more set afloat in the circulation. Thus the poison and the remedy are cast out together by the urine (MELSENS, WILLIAM BUDD). It is necessary, however, to notice the dangerous phenomena which may at first supervene on the administration of *iodide of potassium* in cases of lead poisoning; and great caution is necessary in the employment of this remedy in man for the first few days. At the moment when the metallic compounds fixed in the body become dissolved or transformed, the phenomena of acute poisoning may occur, caused by their liberation. So much is this the case, that the treatment may be supposed to be at first hurtful rather than beneficial. The patient should have beside him a graduated solution of the *iodide of potassium*; and should begin with a small dose (fifteen grains during the twenty-four hours), and afterwards increase or diminish it according to his pains and sensations (MELSENS).

Galvanism, in the form of Faradization, ought to be used as a local stimulant to the nerves, with the precaution that its application is not to be continued too long each time. Ten or fifteen minutes, at three different periods of the day, or of every second day, and persevered in for not less than four weeks, will be found of great service (TODD, ALTHAUS). The beneficial influence will follow, although, in the commencement of the treatment, even a current of very high tension does not cause any movement whatever in the paralyzed muscles. In such cases the beneficial influence seems attributable to the restoration of mobility to the molecules of nerve and muscle by an induced current, and which is necessary to enable them to be physiologically active. Severe shocks, especially in the commencement of the treatment, should therefore be carefully avoided, as by such the weakened excitability of nerve and muscle may be reduced, in place of being fostered and developed (ALTHAUS, *l. c.*, p. 112 and 119). It would be rational, however, to defer the application of galvanism till the lead has been completely eliminated.

#### GOITRE.

LATIN EQ., *Bronchocele*; FRENCH EQ., *Goître*; GERMAN EQ., *Kropf*—Syn., *Struma*; ITALIAN EQ., *Gozzo*.

**Definition.**—*A specific affection of the thyroid gland, induced by the persistent use of water which has percolated through magnesian limestone rocks or strata, and containing the soluble salts of lime in solution.*

**Pathology and Morbid Anatomy.**—The characters of the swelling of the thyroid gland, associated with this morbid state, appear to be different at different stages of its existence. At first the tumor is soft, but it gradually acquires a firm and even a cartilaginous consistence. In the soft condition the cell-elements of the gland seem to secrete a fluid of a thick, ropy, viscid, gelatinous appearance; but





rivulets which flow through the plains in summer, are exempt from the disease; but the residence of a single year at Edmonton, if the river-water of the Saskatchewan is used, is sufficient to render a whole family the subjects of goitre (RICHARDSON). The disease has been known to occur and to affect a family in a very short time, who, being free from the disease while using the *surface-water*, had a well dug, and obtained their water by tapping a limestone rock; and after drinking from this well for a short time the disease appeared among them. There are some waters in a goitrous district in Switzerland, issuing from the hollows of certain rocks, and trickling along crevices in the mountains, the drinking of which will produce *goitre*, or augment goitrous swellings, in eight or ten days, while the inhabitants who avoid these waters are free from the disease (BALLY, WATSON). Dr. Coindet, of Geneva, states that the use of the hard pump-water in the lower streets of that town brings on goitre very speedily; and at Cluses, on the Arne, numerous cretins and goitrous persons are seen in the streets; while lofty cliffs of mountain limestone tower over the town; and through the crevices of these cliffs copious streams of water flow. In Yorkshire, Derbyshire, Nottingham, Hants, and Sussex, in England, where the disease prevails, there is a ridge of magnesian limestone running from north to south through the centre of the district. All along that line goitre prevails to its greatest extent; and, diverging to either side, the disease is found to diminish (INGLIS, *Treatise on English Bronchocele*). The disease has been known to prevail in one great section of the province of Kemaon, in India, south of the Himalayan mountains, and to be almost entirely absent from another section of that district. Both of them agree in their external aspect, altitude, and climatology, but differ so remarkably in their geological formation that an examination of the rocks of the district, into the very villages where the disease abounded, or did not abound, enabled one to predict whether the inhabitants were affected with goitre or not. No instance occurred, in a district extending over 1000 square miles, in which goitre prevailed to any extent where the villages were not situated on or close to limestone rocks (McCLELLAND). Dr. McClelland visited 126 villages scattered promiscuously over an area of upwards of 1000 miles. The following are the results he obtained:

1. Five of these villages were built upon hornblende and mica slate, or on siliceous sandstone, or on green sandstone. They contained 290 inhabitants, not one of whom was a *cretin*, or was affected with *goitre*.

2. Seventy-one of the villages in the same district were built upon clay-slate. These contained 3957 inhabitants; and among them there were twenty-two persons with goitre, or *one* in two hundred of the population. There was not a single cretin.

3. Thirty-five of the villages, having a population of 1160, were built upon Alpine limestone; and in them 390 persons, or more than *one-third* of the inhabitants, had goitre, while thirty-four of them were cretins, or about one person in every thirty-five.

Lastly, goitre is extremely frequent at Secrora, near Lucknow, and in all that district of Oude which stretches towards Nepaul and



shire, where it is called the "Derbyshire Neck." It is met with in some flat situations in Norfolk; and in one village about five miles from Cambridge it is extremely common (WATSON). In South America goitre is met with both in the upper and in the lower course of the Magdalen River, and in the flat high country of Bogota, 6000 feet above the level of the stream (HUMBOLDT). It is also common at the base of the South American Andes. In North America many cases occur in the vicinity of the Blue Ridge, in Virginia. It is prevalent in the mountainous regions of Pennsylvania, New York, New Hampshire, and Vermont (DUNGLISON). In India it prevails in Oude, and along the line of the Himalayan range. It seems to be more common in females than in males, and is rarely seen before the age of puberty; but in districts where the disease abounds, it is on record that children are sometimes born goitrous of goitrous parents (GODELLE, WATSON). The evidence of hereditary transmission, in the strict sense of the term, appears to be doubtful; but predisposition may exist in some, rather than in others, to the development of the disease.

**Cretinism.**—The condition of idiocy named *cretinism* (and associated with goitre in many districts) is of great interest; but the relations of the two are not yet clearly understood. The idiocy of *cretinism* is associated with deformity and imperfection of the bodily organs, the brain, in common with other parts, participating in the imperfection and deformity. (1.) It may be defined as: "*A condition of imperfect development and deformity of the whole body, especially of the head. It is endemic in the valleys of certain mountainous districts, and is attended by feebleness or absence of the mental faculties and special senses; and is often associated with goitre.*" The affection of the mind varies from mere obtuseness of thought and purpose to the most complete obliteration of all intelligence. (2.) Three varieties are to be distinguished: "(a.) *Complete Cretinism*—Synonym, *Incurable Cretinism*,—Cretinism characterized by idiocy, deaf-dumbness, deficiency of general sensibility, and absence of the reproductive power. (b.) *Semi-Cretinism*,—A degree of cretinism in which the mental faculties are limited to the impressions of the senses and the bodily wants; the general sensibility is obtuse, the head is badly formed and drooping, the speech is rudimentary, and the reproductive powers are feeble or absent. (c.) *Incomplete Cretinism*—Synonym, *Curable Cretinism*,—A degree of cretinism in which the mental faculties, though limited, are capable of development; the head is moderately well formed and erect, the special senses, the faculty of speech, and the reproductive powers are present." Dr. Guggenbühl, of Zurich, was the first to recognize the fact that the mental state of *cretins* would be improved by improving the growth and condition of the body. In 1842 he succeeded in buying the mountain of Abendberg, which incloses the plain of Interlaken, and there he established an hospital for these unfortunate children. There the infant *cretins*, removed from the low, close valleys (in which the malady too often finds the circumstances most congenial for its development), are fed and trained in "the free, dry, cool and bracing air of the open but sheltered and sunny slopes of the Abendberg." With but few exceptions, *cretins* are goitrous;



to contain a large quantity of carbonate of lime; whereas the water derived from the clay-slate rock, and which was drunk by the inhabitants who did not suffer from *goitre*, contained none. Such observations as those described, and especially those of McClelland and Greenhow, show that neither the atmosphere, the elevation above the sea-level, the physical aspect of the country, nor locality, have anything to do with the production of *goitre*; but they prove almost to demonstration that the affection is due to some specific action of the drinking-water which flows from rocks of a particular geological formation named magnesian limestone. The circumstances under which these affections were found by McClelland to exist in the low, burning plains of Bengal, formed a striking corroboration to his observations in the hills of Kemaon. *Goitre* and cretinism are very prevalent in different parts of the district of Goruckpore. The soil of the district is of two sorts. One, to which the natives give the name of *Bhat*, characterizes the lands bordering the river Gunduk and its branches. This soil is remarkable for the large proportion of calcareous matter which it contains. One specimen, on analysis, yielded upwards of twenty-five per cent. of carbonate of lime. *Goitre* and cretinism are very prevalent in the villages built upon this soil. In some of them ten per cent. of the population are affected; and of the children in the villages where *goitre* prevails ten per cent. are *cretins*. The dogs and cats of these villages are also often affected with the disease. On the other hand, the lands on the banks of the Gogra consist of a soil to which the natives give the name of *Bangar*. It is much less retentive of moisture than the *Bhat* land, and requires irrigation for the production of winter crops. This *Bangar* soil is very siliceous, and contains scarcely any lime. *Goitre* and cretinism are unknown in the villages built upon this soil (*Brit. and For. Med.-Chir Review*, Jan., 1861).

The natives of Oude ascribe their *goitres* to drinking certain waters; and they adduce cases to prove that by partaking of the water of certain wells they get the disease, and by deserting those wells they sometimes become cured of it (GREENHOW). Thus almost all writers who have written on the subject agree that, in some way or other, the condition of the water has to do with the production of *goitre*. Remarkable instances are known wherein the exchange of well- for rain-water, for drinking purposes, has been followed by the best effects, and even by the disappearance of *goitrous* tumors. Dr. Greenhow states that in Oude, where the water of wells believed to be injurious, in consequence of their excessive impregnation with lime, has been given up, and other water used instead for drinking, great benefit has been felt, and *goitres* have decreased in size, even though the subjects of them have continued living in the same village as before. He was assured also, by several of his patients in Oude, that certain wells were known by them to be deleterious, and that the natives of the villages avoided them accordingly, having learned to do so from experience. He tested the water of the wells most shunned by the natives, and found it to contain a great excess of lime; and he concludes, from his own investigations, in connection with others, that the use of drinking-



may also be employed externally in the form of a liniment or ointment—the *unguentum iodum compositum*, of which a small portion may be rubbed upon the swelling night and morning. In some parts of India the application of an ointment of the *biniodide of mercury* was found very efficacious. The ointment is prepared by adding finely powdered *biniodide of mercury* to melted lard or mutton suet. This ointment is then applied to the goitre about an hour after sunrise; and is rubbed in, by means of an ivory spatula, for about ten minutes—the patient sitting with his goitre held well up to the rays of the sun as long as he can bear the exposure. The ointment will probably produce a blistering effect, although no vesicles appear on the skin; and in the course of the day the ointment should be gently rubbed in again, and the patient sent home, with orders not to touch it with his hands, but to allow the ointments to be gradually absorbed. A second application is sometimes necessary in very bad cases. In 1855 no less than 500 or 600 persons were sometimes treated in a single day; and it is estimated that altogether about 60,000 patients have been so treated; so that the cases in the district are now far less numerous than formerly; and the disorder is thus being extinguished (Mouat, in *Indian Annals* for April, 1857).

When medical treatment fails, surgeons have attempted to give relief to the symptoms by one of three operations; but so long as the disease does not interfere with any of the important functions of the body, nor produce serious discomfort, surgical interference is not warrantable. These operations are,—(1.) The introduction of setons through the tissue of the diseased gland—an operation which has been successful. A thin double wire is to be passed through the gland, and left there for a week (QUADRI, TANNER, JAMES). (2.) Tying the thyroid arteries which supply the goitre with blood, and so starving the tumor, has been attended with varied success (COATES, BRODIE, EARLE, WICKHAM). These means having failed, (3.) The gland has been extirpated—an operation which few surgeons would now think of undertaking.

Dr. Watson justly observes, regarding these surgical interferences for the radical cure of goitre, that “there is not one of which the *average* results have been sufficiently prosperous to warrant its repetition, except in cases where life is put in jeopardy, or made miserable by the swelling; and where other methods, and particularly the treatment by *iodine*, have been tried and have failed” (*Principles and Practice of Physic*, vol. i, p. 795). He makes, however, an exception in favor of puncturing any cell or cyst containing fluid, which sometimes makes up a considerable portion of the tumor. Such cysts may be punctured without much risk, and with great relief to the patient.

The indications for treatment in *exophthalmic goitre* are, to allay the irritability of the stomach by the use of ice; to give bland, unstimulating, nutritious food in small quantities and at short intervals; to produce sound and refreshing sleep by morphia, or any such stimulating soporific; to administer digitalis; to steady the weak heart and control its excitement. Iron may improve the





[Further accounts of this vetch as a cause of paralysis may be found in *Indian Annals of Medical Science*, vol. vii, 1861, by the late Dr. Kinloch Kirk, p. 144; also by Dr. Irving, pp. 127 and 501. An incidental reference is also made in Thomson's *Travels in the Western Himalaya and Tibet*, p. 391, footnote.]

**Symptoms and Phenomena.**—The paralysis is observed most frequently during the rainy season in India—cold and wet being perhaps an exciting cause, so that the first lameness may be a mixture of palsy and rheumatism. Men who had gone to bed quite well awoke in the morning feeling their legs stiff, especially at the knees, their loins weak, and their gait unsteady. Fever does not seem to attend the accession of the more obvious phenomena; but pain gets worse, and eventually the lower limbs become quite paralyzed. The patient walks with difficulty, the toes turn inwards, the legs waste, and the great toe nail scrapes the ground, till, in persons who go barefooted, the nail has been known to get rubbed down to the quick. Males are said to be more often affected than females; and the *Ryots* are more liable to the disease than the *Zemindars*.

**Treatment.**—Some cases seem to have been benefited by generous diet, tonics, the use of strychnine, and of blisters to the loins; but nothing is known definitely on the subject, nor have we any records of the morbid state of the spinal marrow in such cases. Of course, the bad quality of the food must be set right.

---

## CHAPTER XI.

### PATHOLOGY OF THE PARASITIC ORDER OF ZYMOTIC DISEASES.

**The Parasitic** order of diseases are so called from the fact that a great variety of lesions and symptoms of organic disorder are brought about by the presence of *animals* or of *plants* which have found a place to live and subsist within or upon some tissue, organ, or surface of the body of man, or of other animals and plants. The diseases of this order may be considered as due either,—(1.) To the existence of parasites from the *animal kingdom*; or, (2.) To parasites from the *vegetable kingdom*; and all of which live either upon some surface or within a cavity of the body, or within the substance of some of its tissues or organs.

From the animal kingdom we have the *entozoa* and the *epizoa*, and from the vegetable kingdom the parasitic diseases are due to *epiphytes* and *entophytes*. It is only recently that we have been able to point with distinctness to a vegetable parasite finding its way actually into the substance of animal tissues, and there progressing in development. Dr. H. V. Carter, the Professor of Anatomy and

Physiology in the Grand Medical College of Bombay, has described a “*fungus disease*” of the foot, in which numerous minute tubercles, resembling fish-roe, lie beneath the muscles,” and affect the tissues from the bones to the skin (*Trans. of the Med. and Phys. Society of Bombay*).

Plants, as well as man and animals, have their peculiar parasites and parasitic diseases. The mistletoe is a familiar example of a *vegetable parasite*; and the oak apple, or gall-nut, is a familiar example of an *animal parasite* affecting a plant.

It is known, and in many instances it is capable of experimental proof, that some of these parasitic diseases (vegetable as well as animal) may be transmitted or communicated indifferently from animals to man, and from man to animals. The *tape-worms*, the *encysted*, *vesicular*, and *round worms*, are examples of parasites intercommunicable among animals; and *Tinea*, from the “*Dartre tonsurante*” of the horse, ox, and cat, having been communicated from these animals to man, are instances of vegetable parasites intercommunicable among animals. It may be that the blights of plants, or the causes of them, are also communicable to animals and to man. We know that some of the diseases of man and animals are intimately related with famines and unwholesome food, and that famines are due more to diseases of vegetable and animal life than to destruction or loss of food.

The records of history furnish numerous examples of periods of blight in the vegetable kingdom, associated with epizootics among the lower animals, and with epidemics affecting the human family (see Sir William Wilde’s *History of Ireland*, compiled in connection with the census taken ten years ago). The relative connection of these events has scarcely yet attracted the attention of pathologists, human or comparative. Here, indeed, is a wide field for investigation—a territory almost yet unexplored. The medical service of Her Majesty’s British and Indian armies gives golden chances for observation, if the chances are seized at the moment, and the observations connected with the facts already known. To the more salient of these facts the attention of the student is here directed.

Since the beginning of the present century, when Rudolphi published his systematic work on the entozoa (1808), almost every year has contributed new and important facts, which render the subject of *Parasitic diseases* one of increasing interest to the pathologist and the physician. The subject abounds with most puzzling riddles in natural history and pathology, especially concerning the reproduction, the development, and the propagation of parasites. So long as 170 years ago (1691), the independent nature of such structures as the “*hydatid cyst*” was established by Tyson (*Phil. Trans.*, cxiii, p. 506); and it was stated by Pallas in 1766 that all the cystic worms were forms of tape-worms belonging to one species—namely, the cystic or hydatid tape-worm; but it was not then known how their generation and propagation was effected. For a very long time the received doctrines regarding the generation and de-

velopment of living beings were tacitly set aside in behalf of such "existences." They were believed to arise spontaneously. Inquiry was thus set at rest, curiosity seemed satisfied, or investigations followed a fruitless direction—as when observations were made on such cysts, in the hope of discovering in them some evidence of the existence of organs of generation, or evidence of some process of generation analogous to what prevails in other animals. Ova were looked for, and organs of generation were looked for, where neither ova nor organs of generation existed. The calcareous particles visible in the tissues of those animals were at one time mistaken for eggs, and described as such, in the membrane of the *Cysticercus* (1841). At last, in 1842, a great insight was obtained regarding the nature of the generation and development of these and other parasites by the publication of facts which showed that amongst a certain class of minute *Cercariæ* (worms of a microscopic size found in stagnant water), the generation of them was carried on through a series of broods produced from one parent, each brood differing from the parent and from each other. The discovery of this fact was due to Steenstrup. He described the phenomena under the name of "alternation of generation" amongst these *Cercariæ* which ultimately live within the body of different mollusca (*Planorbis* and *Lymnæus*).

These observations gave quite a new direction and impetus to investigation; and Steenstrup himself foretold that the hydatid cysts would be proved to be undeveloped tape-worms, each cyst capable of producing a tape-worm after its kind.

This view was at once taken up, and independently worked out by Eschricht, Nordmann, Von Siebold, Kuchenmeister, Kræmar, Zenker, Leuckart, Weinland, in Germany; Von Benedin, in Belgium; Dujardin, Blanchard, and Robin, in France.

Many physiologists and physicians of this country have been no less accurate observers. Barker, Bristowe, Nelson, Erasmus Wilson, Gulliver, Gull, Jenner, Busk, Rainy, Cobbold, and Bastian, may be particularly noticed; and many valuable records have been published in isolated papers by officers of the Army Medical Department.

The conjoint researches of these extensive workers have found most philosophical expositors in this country in Dr. E. A. Parkes, the Emeritus Professor of Clinical Medicine in University College, and now Professor of Hygiene in the Army Medical School (*Brit. and For. Med. Review*, 1853); in Dr. Allen Thomson, Professor of Anatomy in the University of Glasgow (*Glasgow Med. Journal*, No. x, July, 1855); and lastly, in Dr. William Brinton, in the *Brit. and For. Med.-Chir. Review* for 1857. From these and many other later sources the following concise account may be given relative to the parasitic order of diseases, and their rational treatment.

Kuchenmeister and Von Siebold were the first to prove by experiment that the hydatid or vesicular worms were the young or larval states of tape-worms; and they demonstrated—(1.) That each parasite had an independent life of its own. (2.) That most animals have each their own peculiar parasites—that even parasitic

animals are themselves infested with parasites—an observation embodied in the Hudibrasian couplet,—

“These fleas have other fleas to bite 'em,  
And these fleas, fleas, *ad infinitum*.”

The experiments of Kuchenmeister and Von Siebold further demonstrate—(3.) That some parasites pass or migrate from the body of one animal into that of another (including man), or from one part of the same animal to another cavity or viscus in it; such migrations being required for the introduction of the entozoa or their ova into the animals they inhabit, and where they undergo those series of changes about to be described, by which they reach maturity. (4.) That thus, through food or drink, or both, entozoa pass into the human body, finding their way into the most delicate tissues, as most minute ova or embryos. (5.) That they undergo progressive changes of development towards maturity in each of the new localities where they find subsistence and protection.

We cannot now rest satisfied with a mere knowledge of the general appearance of these so-called “worms” as they are found in man and animals. It behooves the physician to ascertain their origin, their source, and their mode of entrance into the body they inhabit. The easy but unsatisfactory hypothesis of “spontaneous generation” can no longer be entertained. On the contrary, it is now clearly established that all the parasitic entozoa are produced more or less directly from fecundated ova. The general and minute anatomy of these “worms” must be studied, as well as their modes of generation, of reproduction, and phases of progressive development, the various metamorphoses of their individual forms, and their transigrations from one animal into another. We must become acquainted with their existence even in plants, as well as in animals, and in other animals besides man, especially in such animals or plants as constitute the food of man—fish, flesh, fowl, mollusca, and crustacea,—and especially all fresh-water plants, or plants which grow on moist ground.

A knowledge of details relative to generation and reproduction is absolutely necessary in order to appreciate the nature of parasitic diseases. Indeed, without such knowledge no advance is likely to be made in the prevention of these diseases. It is this kind of knowledge which has recently led to most important practical results in the history of animal parasites, and which most of all seems capable of extending the science of parasitic diseases, especially in relation to human pathology, and the rational treatment of such diseases.

Parasites of animal organization exist in man and animals in every grade of development; and the first lesson for the student to learn is, how to distinguish entozoa which are sexually complete from those parasitical productions which are destitute of sexual organs, but which have long been regarded as distinct animals.

At least thirty well-marked forms of entozoa have been described as infesting the body of man. They may be enumerated in a classified list as follows:

**\*LIST OF GENERA AND SPECIES OF HELMINTHOID ENTOZOA WHICH HAVE BEEN DISCOVERED INFESTING THE HUMAN BODY.**

**A. SOLID WORMS: PLATYLMIA; vel, STERELMINTHA.**

**I. CESTOIDEA—Banded, riband-like, girdled, or tape-worms in the form of—**

**1. Mature sexual parasites, androgynous, and living in the alimentary canal.**

(a.) *Tæniæ*. 1. *Tænia solium* (LINNÆUS), the common tape-worm of man in this country.

2. " *mediocanellata* (KUCHENMEISTER), the common tape-worm of man on the Continent.

3. " *marginata* (BATSCH, COBBOLD).

4. " *elliptica* (BATSCH, COBBOLD).

5. " *acanthotrias* (WEINLAND, COBBOLD), its larva, scolex, or cysticercus only known.

6. " *nana* (SIEBOLD), a very small filiform *Tænia*.

7. " *flavopunctata* (WEINLAND, COBBOLD).

8. " *echinococcus* (SIEBOLD).

(b.) *Bothriocephali*. 1. *Bothriocephalus latus* (BREMSE); vel, *T. lata* (LINNÆUS), the broad tape-worm, endemic to man in some localities only. Its embryo is ciliated and developed in water (KNOCH).

2. *Bothriocephalus cordatus* (LEUCKART), new to science; recently found in North Greenland.

**†2. Immature non-sexual, cystic, or vesicular parasites, the embryonic form of the genera sub (a.) *Tæniæ*.**

(a.) *Cysticerci*. 1. *Cysticercus tæniæ cellulosa* (RUDOLPHI), the larva or scolex of the *T. solium*.

2. " *tæniæ medicanellata* (LEUCKART), the larva or scolex of *T. medicanellata*.

3. " *tenuicollis* (RUDOLPHI), the larva of *T. marginata*.

4. " *tæniæ elliptica*, at present unknown.

5. " *tæniæ acanthotrias* (WEINLAND), only the cysticercus found; mature *Tænia* not yet found.

6. " *tæniæ nanæ*, at present unknown; probably inhabits insects (LEUCKART).

7. " *tæniæ flavopunctata*, also at present unknown.

(b.) *Echinococci*. 8. *Echinococcus hominis* (RUDOLPHI), the larva of *Tænia echinococcus*,

**II. TREMATODA—Fluke-like parasites.**

1. *Fasciola hepatica* (LINNÆUS); vel, *Distoma hepaticum* (RUDOLPHI).

2. *Distoma crassum* (BUSK); *Distoma Buskii* (LANKESTER).

3. *Distoma lanceolatum* (MEHLIS).

4. *Distoma ophthalmobium* (DIESING).

5. *Distoma heterophyes* (SIEBOLD).

6. *Bilharzia hæmatobia* (COBBOLD); vel, *Gynæcophorus hæmatobius* (DIESING).

7. *Tetrastoma renale* (DELLA CHIAJE).

8. *Hexathyridium pingucola* (TREUTLER).

9. *Hexathyridium venarum* (TREUTLER).

**B. HOLLOW WORMS: NEMATELMIA; vel, CIRCLELMINTHA.**

**I. ASCARIDES—Unisexual, body attenuated posteriorly, and still more so anteriorly, mouth with three tubercles, tail of the male narrower than that of the female.**

1. *Ascaris lumbricoides* (LINNÆUS).

2. *Ascaris mystax* (RUDOLPHI, COBBOLD); vel, *Ascaris alata* (BELLINGHAM).

3. *Trichocephalus dispar* (RUDOLPHI).

\* The individual names in this list are similar to those published by Dr. Cobbold in a number of the *Lancet*, before the publication of the second edition of this work. In that list a number of forms were introduced for the first time by Dr. Cobbold, which had never before been noticed by any of our systematic writers; and Dr. Cobbold took great pains in working out this point. His name, therefore, appears to identify them in the list. (See also his great work *On Parasites*, recently published.)

† N.B.—The appearance produced in the flesh of animals by the growth of these vesicular parasites has been named "the measles," or "measly flesh."





from the ovum have now been actually observed in a considerable number of the parasitic entozoa; and it is to be remembered, as a general fact, that the development of the ova rarely takes place in the same animal, or in the same part of an animal, where the parasitic entozoön has passed its life and has exercised the generative function. There is either a migration from a parasitic to a free condition for a time (e. g., *Guinea worm*, *Ascarides*, *Cercariæ*); or from one animal into another animal, the free condition intervening (e. g., *Bothriocephalus*); or, lastly, the migration may take place from one part to another of the same animal who is the unfortunate host (e. g., *T. spiralis*; and cases of tape-worm giving rise to *Cysticercus*). Some entozoa, known only as incomplete or immature animals in the parasitic mode of life, attain to sexual maturity in the free state; others again, and perhaps the greater number, after living free for a time, become sexually complete in the parasitic condition (e. g., the *Ascarides* and the *Bothriocephalus*).

“The migrations or changes of habitation of the entozoa, or their ova or embryos, appear to take place in a variety of ways: *first*, by their being passed out of the body of the inhabited animal with the fæces or other secretions; *second*, by their being introduced into the bodies of inhabited animals with their food or drink; *third*, by their directly piercing the integument or other tissues, *fourth*, by their piercing the membranes and parenchyma, entering the bloodvessels, being distributed through them, and subsequently piercing their coats to attain other situations.

“Some of these entozoa are directly developed from their ova, without undergoing more remarkable changes than those which are known usually to accompany the process of embryonic evolution in many other animals. Other entozoa are subject to individual metamorphoses, or the embryo passes through successive stages of development, of so remarkable a character as to mask the regular sequence of the phenomena of progressive formation. There are others of the entozoa which are subject to still greater changes in the progress of their existence,—changes upon which great light has recently been thrown by the remarkable researches of Steenstrup and others, in regard to what has been called alternate generation or *metagenesis*. Thus some of the entozoa, by a non-sexual process, undergo that peculiar form of multiplication in which the immediate progeny of development from the ovum is dissimilar from the parent, but produces, without the aid of sexual organs, another progeny, which either itself, or by repetition of an analogous process, returns to the parental form. This is a process of the nature of an internal or external gemmation, which is often attended with a prodigious multiplication of the number of individuals. In some entozoa, again, metamorphosis and metagenesis are combined. It is obvious that the external conditions necessary to maintain these varieties of the vital states must be different” (Allen Thomson, *Glasgow Med. Journal*, l. c.).

The lesions and diseases caused by the existence of parasites rather tend to embitter existence than to cause death; and they are especially frequent amongst soldiers. With one exception—namely, in the case of the immature cystic parasites—the disorders





vermination are connected, not with their direct irritation of the mucous membrane with which they are in relation, but with a general disorder of the system, partly resulting from the parasites, and partly the cause of their maintenance and development in the intestinal tract."

## CHAPTER XII.

### DETAILED DESCRIPTION OF THE PARASITES, AND OF THE LESIONS ASSOCIATED WITH THEM.

#### SECTION I.—THE ENTOZOA.

##### TAPE-WORMS—*Cestoiden*.

**Definition.**—*In their mature condition the tape-worms are all more or less jointed entozoa, of a riband-like form, marked with bands, or girdled. Each mature joint or segment is of hermaphrodite conformation, containing at once male and female reproductive organs, which produce fecundated ova. In their immature condition the embryo penetrates the tissues, and becomes encysted. In this stage of development they are known as the "cystic entozoa."*

**Pathology.**—Eight varieties of true tape-worms have been found in man, and two varieties of the *Bothriocephalus*. But two only of the true tape-worms are of frequent occurrence—namely, the *T. solium* and the *T. mediocanellata*. The former is the one endemic in this country; the latter is the more common tape-worm on the Continent and in South Africa.

The *Bothriocephalus latus* is endemic in some well-defined localities, chiefly in continental Europe; and the *B. cordatus* is new to science, having been but recently found in North Greenland (LEUCKART). These tape-worms have been known for a very long period; but they have not always been distinguished from each other. Indeed, the distinguishing characters are but recently known. They have often been confounded together under the name of "solitary worm," because it was believed they lived singly. This, however, is a mistake.

The *T. solium* and *T. mediocanellata* appear at first sight to be very similar to each other in general appearance. The latter is much the larger of the two. It is only in the alimentary canal—the small intestine of man—that they become sexually mature, in natives of France, Italy, Holland, Germany, and Great Britain. The *Tænia* has been also found in Egypt, and is very common in natives of Abyssinia;—so common is it there, that its absence is the exception to the rule. The affection is there looked upon as a natural occurrence; and so general is this belief, that when a slave is sold into Abyssinia he provides himself with a plentiful supply of kousoo—the local remedy for expelling the parasite.



at different parts of the body. They are square or oblong; and in the mature part of the animal the length of them is equal to twice the width. The anterior border of each segment unites with the anterior or previous segment, and is thinner than the posterior border, and also narrower. The posterior border is thick, and projects or overlaps the border of the segment next in order, and is undulating or indented. The lateral margins incline to each other anteriorly. The two surfaces are flat or slightly elevated towards the centre.

Each mature segment contains male and female organs of generation. The opening at the side of each segment is the sexual aperture, indicated by a prominent papilla, once supposed to be a sucker. These openings are sometimes at one side and sometimes at the other. Two, three, or four consecutive segments may have them on the same side, or on opposite sides; but there is no regular alternation. With a lens a cup-shaped depression may be seen, showing two mesial apertures. From one of these a *lemniscus* or rudimental penis projects, connected with a horizontal (deferent) canal (sometimes indicated by dark pigmentary material) from a vesicular body in the middle of the posterior end of the segment (OWEN).

Behind this male orifice is the opening to the female organs, by a canal leading to a lobulated organ, which is the ovary or germ-stock. These parts are more distinctly developed the farther the segments examined are from the head end of the worm. While the head continues to adhere, by its circles of hooklets and oscula, to the mucous membrane of the intestine, the last or caudal joints, when they have arrived at sexual maturity, are separated one by one, or

FIG. 6.\*

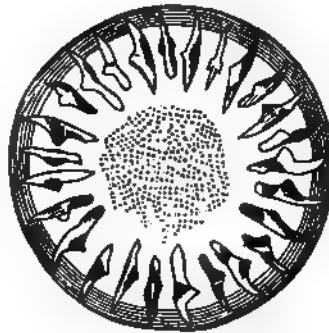


FIG. 7.†

\* Circle of hooks more highly magnified (after LEUCKART).

† Proglottis of *Tenia solium* magnified.—(a.) Genital pore, with its preputial cover or sheath-skin; (b.) Lemniscus or penis; (c.) The oviduct; (d.) The seed-vessel; (e.) The uterus; (f.) The water vascular system of vessels (after ROKITSKY).

in numbers together, and new joints are at the same time gradually formed behind the head. Thus growth and development take place mainly towards the neck of the parasite, by a process of transverse fission; and thus a segmented individual or compound animal appears to grow. This segmentation of individual links by transverse fission ceases when the organs of generation begin to develop themselves in them; and when those are complete, the segment or link has arrived at sexual maturity or completeness. It is now called a *proglottis*. Thus all the new segments come to be developed between the head and those which are advancing to sexual completeness; and if the characters of complete sexual development be taken as the distinctive mark of individuality, then each segment of the tape-worm may be looked upon as a distinct animal; and this separation by fission or segmentation may be considered as analogous to what takes place in the medusæ or polypes—a kind of alternate generation, in which the *segments*, *zoönites*, or *proglottides* may be regarded as making up a colony of animals. It is only in the alimentary canal of man and other animals that the tape-worms, or cestoid entozoa, attain to sexual maturity; and in all of them the ova are fecundated before being discharged, and may often in the *T. solium* be perceived to have undergone the first stage of their development before they are excluded from the oviduct of the mature segment. The expulsion of the ova occurs in some one of the following ways:

FIG. 8.\*



(1.) The impregnated segments separate from each other, and passing out of the body singly or in numbers with the faeces, or without any fecal evacuation, become decomposed, and so the eggs are set at liberty. The activity of these separate segments is retained for a considerable time after passing out of the body—a circumstance which led to their being at one time taken for a dis-

tinct species of worm, to which the name of *Vermes cucurbitini* (from resemblance to a pumpkin-seed) was applied. The contracted appearances of a segment during its movements out of the body are represented by the forms shown in the accompanying woodcut (Fig. 8).

One may readily observe the activity displayed by these *beauties of nature* as they disport themselves on the recently extruded excrement of almost every constipated dog. The expelled joints may be seen to become violently contracted shortly after their expulsion, as if the stimulus of physical climate in their new situation provoked excessive contractions. The long single joints thus expelled become still more elongated by contractions of their transverse fibres, while the alternate contractions of these fibres with the

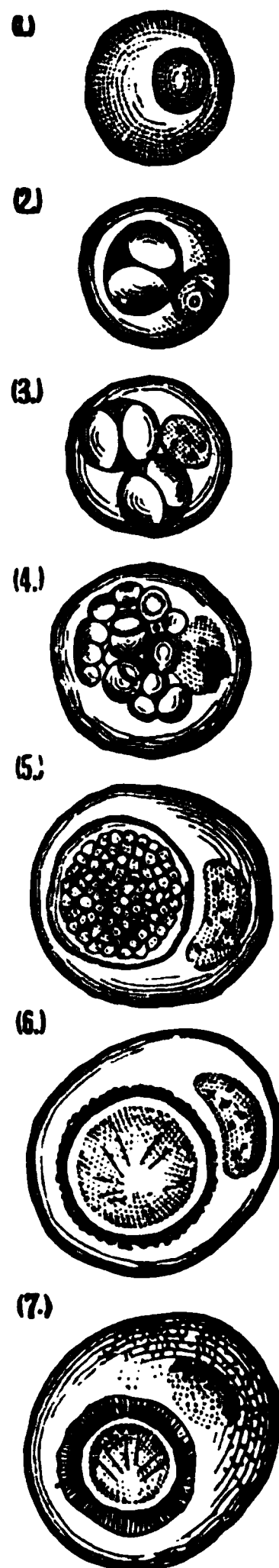
\* Proglottides of a *Tenia (mediocanellata)* in various stages of contraction (after LEUCKART).

longitudinal ones cause shortening of the joint to such an extent that its breadth exceeds its length. Such a sequence of contractions produces movements which simulate those of progression in a worm, and thus these segments may be seen to move some little distance from the spot on which they may have first fallen, discharging ova during their march from the interior of the segment. (2.) Eggs are thus discharged through the genital pores of the mature segments; and if the segment be slightly squeezed, the ova may be pressed out. Such a contingency is not unlikely to happen within the rectum, when, by constipation or otherwise, the matured joints are retained, and constitutes one of the most serious dangers which the matured tape-worm inflicts on the animal it inhabits, and one of the strongest indications for its removal. It has been recently ascertained that in one or two instances the presence of a *Cysticercus cellulosæ* (the embryo of the *T. solium*) has been found to coexist with the previous prolonged existence of a *T. solium* in the intestinal canal of the human subject. (3.) The mature joints of the adult tape-worm seem, in some instances, to undergo a disintegration within the intestine of the animal they live in. Thus, Kuchenmeister on one occasion found the wall of the large intestine of a dog occupied by a white sandy powder, the particles of which, on examination under the microscope, turned out to be innumerable ova of a *T. serrata* which lived higher up the bowel, accompanied by its separated joints.

The mature segments are often expelled from the human rectum at the rate of six or eight a day, and they exhibit evidence of very active vitality for some time. Moisture is favorable for maintaining their existence, and for favoring the spread of the eggs over herbs, grass, ground fruit, or vegetables, which may become the food of man or of cattle.

The structure of these ova (Fig. 9) is peculiar; and the provisions possessed by their coverings for preserving the embryo are important points for consideration in connection with their transmissions through apparently impossible conditions into the bodies of animals, where they become further developed; and in connection with their powers of resistance to therapeutic agents (which have been called anthelmintics or vermifuges) administered for their removal.

FIG. 9.\*



\* Development of the ovum of *Tania solium*.—(1.) Previous to segmentation; (2, 3, 4, 5.) Segmentation in the impregnated ovum; (6.) Appearance of the early embryo, with its three pairs of siliceous spikelets; (7.) Mature condition of the ovum containing the embryo inclosed within its leathery case (after LEUCKART).



shaped stiletto. The lateral pairs of these spikelets are then brought backwards to a rectangular position, and so they thrust the embryo forwards in the direction in which the anterior pair of spikelets pointed. Similarly repeated actions eventually accomplish progression to a resting-place; and the action may be aptly compared to the movements of the arms and attitude of the head of a swimmer. But this active migration is not the sole means by which the embryo *Tænia* is enabled to traverse the animal body. The embryo may penetrate a mesenteric vein, when it will at once be swept onwards by the current of the blood to the portal vein, and passing into the minute ramifications of the portal system, may find a resting-place in the liver. Leuckart has found the embryos of tape-worms in the blood in such large numbers that he inclines to regard the currents of blood in the vessels as the ordinary and more usual channels for the migration of the embryos. It also explains the wide diffusion of *tape-worm* embryos as *cysticerci* or *echinococci* in various stages of development throughout different viscera of the body, where they become encysted, and especially their very frequent site in the liver, peritoneum, and mesentery. Thus far completed and encysted, the embryo is called a "*scolex*." The embryos of *Echinococci* and *Cænuri* give rise to numerous *scolices*, which complete their development into tape-worms in the alimentary canal of another animal, when that animal happens to eat the liver or brain containing the cysts of such *Echinococci* or *Cænuri*; but the embryos of such *echinococci* or *cænuri* tape-worms find their way into man or animals with drinking-water, or with raw, uncooked articles of vegetable diet from moist soils, such as salads, roots, fallen fruit, all of which may be doubtless so exposed as to receive the germs or ova containing the embryos, passed along with fecal excrement of dogs especially, and which, after being dried, are carried by wind or water in all directions.

The third stage of development consists in the formation of segments, which are first seen in the form of marks, like girdles, surrounding that portion of the entozoön next to its oscula and hooklets, and which terminate in a caudal vesicle. It is now an incomplete segmented *Tænia*, and in scientific nomenclature is called a *Strobila*; and the development to this stage may occur while the entozoön is still within the closed cyst which has formed round it.

It is only in the alimentary canal of animals that the last and perfect stage of development is attained, by the tape-worm reaching sexual maturity. The segments or links marked off by the bands, joints, or girdles in the encysted *Strobila embryo* become mature segments by the development of sexual organs within them. This only takes place after the *Strobila embryo* has passed into the alimentary canal of an animal which can afford it a place to live and spend the rest of his days as a fixture attached by its hooks to the mucous membrane. The human alimentary canal is an oft-chosen place of the *T. solium* and *T. mediocanellata*. Here the tape-worm forms complete sexual segments or links, each being hermaphrodite, and tending to separate when completely mature.

After living for some time in this prolific condition, and having





valid soldiers who died at Fort Pitt, and at the Royal Victoria Hospital at Netley. In one instance three very large and long worms existed in the small intestines, each of them precisely similar in all respects. The soldier in whose intestine they were found died of *diabetes mellitus*, and he had been a cook for many years to a military mess at the Cape of Good Hope; another case was that of a soldier who had been also a long time at the Cape.

3. The *Tænia marginata*, produced from the *Cysticercus tenuicollis*, is only as yet known to infest man in its immature state as a *cysticercus*. The full-grown tape-worm being found in the dog and wolf, it is often confounded with the *T. serrata*, from which it differs in its comparatively bulky size and the peculiar form of its hooks. The proglottides nearly equal in size those of the *T. solium*. In its scolex or immature condition this parasite has a very wide distribution; for, in addition to its occasional presence in man, it has likewise been found in various monkeys, in cattle and sheep, reindeer, and in many other ruminants; in horses, swine, and even in squirrels. Its *habitat* is for the most part the peritoneum (ROSE and others). The *cysticerci* occasionally attain an enormous size.

4. The *Tænia elliptica*, whose *cysticercus*, or embryotic condition, is not yet known, is common to cats and dogs, and is known to infest man (ESCHRICHT, LEUCKART). Weinland believes that the *cysticercus* will be found in flies, and that dogs obtain the larvæ by snapping at dipterous insects.

5. *Tænia acanthotrias*, like the *T. marginata*, is only known in man as an embryo or *cysticercus*. From twelve to fifteen of them were found in the muscles of a woman about fifty years of age, by Dr. Jeffries Wyman in 1845. The woman was a subject in the dissecting-room at Richmond, United States. The rostellum of this parasite is furnished with three rows of hooks, fourteen in each row.

6. *Tænia nana*, when fully grown, attains a length of eight or ten lines, and carries from 150 to 170 joints. Its hooks are essentially the same in form as those of other *Tænia*, only they are very minute, and have a peculiar form, owing to the close approximation of the claw and of the anterior root-process (LEUCKART), which gives them a "bifid" appearance. Its head is comparatively large and obtuse, with a long neck. It was first described by Bilharz in 1851, having been found in Egypt in the intestine of a young man.

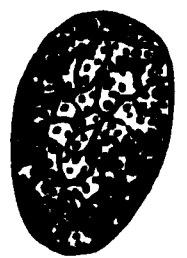
7. *Tænia flavopunctata* measures about eight to twelve inches long. The proglottides are short, and there is a yellowish spot, clearly visible to the naked eye, situated about the middle of each joint, which reminds one of the color and situation of the genital organs as seen in the *Bothriocephalus*. The reproductive orifices occur all along one side of the worm, and the eggs are unusually large. Only one instance of the occurrence of this parasite is on record: it was obtained in considerable numbers by Dr. Ezra Palmer in Massachusetts, in 1842, from an infant nineteen months old. They were expelled without medicine, their presence not having been suspected (WEINLAND, COBBOLD).

8. The *Tænia echinococcus* is very often seen in Iceland, where, in



mature segments a very marked appearance. The neck is not always obvious, for the worm has the power of making it long and thin or thick and short; and there are no joints or segments to be seen in it, but merely prominent ridges. The segments, when they become first apparent, are nearly square; but afterwards they become much wider than they are long. There are two orifices on one of the flat surfaces of each segment; the anterior orifice is connected with a male organ of generation, the posterior is connected with the female. The proglottides are never passed singly, but always in chains of many links, and particularly in February, March, October, and November. The ova (Fig. 12) are always discoverable in the fæces, of an ovoid form, with a perfectly translucent operculated capsule, through which the segmented yolk is distinctly visible; and at the period of discharge of the proglottides the ova show merely the stage of segmentation of the yolk. The *six-hooked* embryo, cased in a mantle studded with vibratory cilia, develops itself after segmentation, protected by the capsule in fresh water, for several months after the expulsion of the proglottides. When so far matured, the lid of the capsule opens up, and the ciliated embryo escapes (Fig. 12*a*), and becomes globular in shape, and moves actively about for a considerable period (a week). If during this period they do not succeed in obtaining access to the intestine of an animal adapted for their development, they lose the ciliated mantle, and perish. When these embryos are introduced by experiment into the intestines of mammals, the *scolices* and mature *Bothriocephalus* were found. Experiments in which living embryos were introduced by implantation between the brain and dura mater, and into the eyes of dogs, also under the skin of frogs, and by injection into the bloodvessels of mammals, give a negative result; *quoad* the development into *cysticerci* or *scolices*. So, also, feeding experiments with the *scolices* of the *Bothriocephalus* found in various fish lead to negative results; just as the feeding of fish with the eggs themselves. It is therefore justifiable to assume that drinking-water from lakes and rivers is the medium through which the living embryos of the *Bothriocephalus latus* find their way into the intestines of men and of mammals (Dr. J. Knoch, *Petersburger Medicinische Zeitschrift*, 1861; COBBOLD, *l. c.*).

FIG. 12.

FIG. 12*a*.

10. *Bothriocephalus cordatus*.—This species (Fig. 13, *a*) has only very recently been described by Leuckart, who received about twenty specimens from Godhaven, in North Greenland, one of which was from the human intestine. The parasite measures about a foot in length, and exists in dogs in considerable abundance. It differs from *Bothriocephalus latus* in the form of the head, which is heart-shaped (Fig. 13, *b* and *b'*), or obcordate, short, and broad, and set on to the body without the intervention of a long neck. The segments are distinct from the very commencement, near the head; and so rapidly do they increase in width, that the anterior end of the body becomes lancet-shaped. About fifty joints are immature; and in the largest example Leuckart counted a total of 660 joints. It dis-



the heart, liver, choroid plexus, the brain, in the tissue between the sclerotic and the conjunctiva, in the anterior and posterior chambers of the eye (MACKENZIE), and in the retina (GRAEFE). The head resembles that of the *T. solium*, and carries thirty-two hooks in two rows, and the neck varies greatly in length. The parasite is especially frequent in domestic swine, and in them it produces the appearance known as the "measles," or "measly pork."

2. The *Cysticercus ex tænia mediocanellata* is to be found in the muscles and internal organs of cattle. For our knowledge of the larval state of the *T. mediocanellata* we are mainly indebted to Professor Leuckart, of Giessen. He has artificially reared them in the flesh of calves, from the eggs of a *T. mediocanellata*; and recent experimental researches incontestably prove that the "measles" of cattle give rise to the *T. mediocanellata*. He fed two calves with the fresh eggs of the *T. mediocanellata*, by giving them the *proglottides* of this parasite. The first animal he experimented on died from a violent attack of the measles disease; and on dissection the muscles were found filled with measles, or vesicles containing imperfectly developed *scolices*. On the second occasion a smaller number of *proglottides* (in all about fifty) were administered, and the febrile symptoms again appeared with such virulence that Leuckart thought this animal would die also. Fortunately, after the lapse of a fortnight from the commencement of symptoms, some abatement of the disease took place, and this gradually continued until the animal was perfectly restored to health. Eight and forty days subsequent to the earliest feeding experiments (which were continued at intervals for eighteen days) Professor Leuckart extirpated the left cleido-mastoid muscle of the calf, and whilst performing the operation he had the satisfaction of seeing the *cysticercus* vesicles lodged within the muscles. They were larger and more opalescent than those of the *Cysticercus tænia cellulosæ*, but nevertheless permitted the recognition of the young worms through their semi-transparent coverings. The heads of the contained *cysticerci* exhibited all the distinctive peculiarities presented by the head of the adult *Strobila* (the *T. mediocanellata*). Taking the results of this experiment in connection with previously ascertained facts, the most unequivocal evidence is brought together that man becomes infested with the *T. mediocanellata* by eating imperfectly cooked veal or beef in which the *cysticerci* abound.

3. The *Cysticercus tenuicollis* is rarely found in man, but it has occasionally been found in the mesentery and in the liver. Eschricht and Schleissner have shown that these *Cysticerci* are sometimes associated with the *Echinococcus* in Iceland (COBBOLD, *l. c.*).

4, 5, 6, and 7 require no special notice.

8. The *Echinococcus hominis* is the larva of the *T. echinococcus*; and the first accurate description of the immature form of the parasite was published by Bremser in 1821. These parasites have been and are still often indifferently named "hydatids" or "echinococcus cysts;" but English writers have restricted the term "*hydatid*" to designate the enveloping cyst, and the term "*echinococcus*" to signify the contained entozoön. The *Echinococcus* is an extremely common



merous large and small vesicles, more or less clear and transparent, floating free, or so closely packed together that they cannot be removed without some degree of pressure. Some of them, particularly the smallest, adhere to the germinal membrane. They vary

FIG. 14.\*

(1.)

(2.)

in size from that of a millet-seed to a size as large as a goose's egg, and their number not unfrequently amounts to several hundreds (560, PEMBERTON), or even thousands (7000 and 8000, ALLEN, quoted by PLOUCQUET and FRERICHs). The larger of these free vesicles sometimes contain smaller ones of a third generation, and occasionally they in their turn contain others of a fourth generation. The size of the "hydatid tumor" and the germinal membrane must increase and grow according to the number and size of the daughter vesicles, and in proportion to the quantity of contained fluid, which is sometimes rendered slightly opaque by the quantity of embryo *Echinococci* floating free in it. From the rotundity and distension of these inclosed vesicles it is difficult to fix them for examination; and when they are punctured, their fluid contents issue from the vesicle in a jet of considerable force, impelled by the contractile power of the elastic tissue; and if the incision be of a sufficient size, the vesicle will roll up, and turn itself inside out. The *Echinococcus* embryo varies in size from  $\frac{1}{8}$ th to  $\frac{1}{4}$ th of a line in length in the contracted state, and from  $\frac{1}{8}$ th to  $\frac{1}{4}$ th of a line in its elongated form. These variations in length are according as the head of the parasite is extruded or retracted within the vesicle. The contracted state, in which the head is retracted within the vesicle, is the form most commonly seen in the "hydatid tumor," after removal from the organ in which it was developed. In this state it is usually globular or oval, and slightly flattened at the opposite poles. In the elongated state, when the

FIG. 15.†

\* Fig. 14.—Groups of *Echinococci*, showing—(1.) The pedunculated connection between these parasites and the germinal membrane; (2.) Their occurrence in groups, enveloped by a very delicately thin membrane, continuous with the germinal membrane (after ERASMUS WILSON).

† Fig. 15.—Two *Echinococci* from a "hydatid tumor." The one has the head retracted within the vesicle; the other has the head extruded.





is the resistance opposed to the further growth of the *Echinococci*, which may even be thus arrested, so that the parasites die, and a spontaneous cure results (CRUVEILHIER, FRERICHS.)

Compound "hydatid tumors" have been found in which the cavity is multilocular. Outgrowths or buds form, which give an alveolar character to the lesion (BUHL, VIRCHOW, FRERICHS).

It has been usual to consider that there are two distinct forms of *Echinococci*, severally referable to different tape-worms; but they are now regarded as one and the same. The *Echinococci* so prevalent in Iceland are known to occur indifferently in men and oxen; and are sometimes so prevalent that about one-eighth of all the cases of disease are referable to this cause; and generally several members of one family suffer (LEARED).

**Acephalocysts** were first described by Laennec as growths of membranous cysts. In the present state of science they are regarded as abortive *Cysticerci* or *Echinococci*—parasites of one or other of those kinds in which the development has been arrested. Remains of hooklets have been found in them; and the structure of the cyst may sometimes be seen to be precisely similar to that of the *Echinococcus* cyst. They have been found in all those places where the *Cysticerci* and *Echinococcus* cysts are known to abound—*e. g.*, liver, spleen, kidney, bladder, and in the exostoses of bones.

Thus we have seen that each kind of *Tænia* has not only its own definite vesicular embryo, but each *Tænia* has a definite *Cysticercus* or *Echinococcus*; and all of them are capable of being developed or reared into *Tænia* when transferred into the alimentary canal of a suitable animal; and conversely, the development of *Cysticercus* and *Echinococci* occurs in man and other animals in consequence of segments of tape-worms, or the ripe ova they contain, being eaten with their food.

The experimental proof of these statements it is the principal scientific merit of Kuchenmeister to have established, by experiments which date so far back as 1851; and they have since been repeated and confirmed by many other observers (SIEBOLD, NELSON, ZENKER, LEUCKART, WEINLAND, KNOCH).

The experiments of Kuchenmeister were made on a variety of animals, and in one instance on the human body. The following is a short summary of his observations, together with those of Von Siebold and others, condensed from Dr. Allen Thomson's valuable paper in the *Glasgow Medical Journal*, No. x, July, 1855, and which demonstrates the

**Relation between the Cystic and the Cestoid Entozoa.**—These experiments mainly consist in observing the effects of feeding an animal with the ova or larvæ with which it is designed to affect it. The first experiment of this kind was performed by Kuchenmeister in 1851. He caused young dogs to eat with their food a number of the *Cysticercus pisiformis* of the rabbit and hare, and found that after some weeks these *Cysticerci* were converted, in the intestine of the dogs, into the *T. serrata*.

The more important of the experiments devised and performed



thirty-eight days the *Tæniæ* had arrived at maturity, and appeared, like those in the previous experiments, to correspond exactly with *T. serrata* and *T. solium*; in two other dogs the experiment was rendered nugatory by the dogs being ill of distemper at the time.

*Fifth Series.*—The last of the experiments related by Von Siebold were made with the *Echinococcus* animalcules of domestic cattle, which are probably not specifically different from those of man. As many as twelve young dogs, and also a fox, received a quantity of the small *Echinococci* in milk; and on being examined at various periods from the commencement up to twenty-six days, there were found, in all different stages of development, small *Tæniæ* totally different from any observed in the previous experiments, or indeed from any accurately distinguished or described by helminthologists. Von Siebold proposes to call this variety *T. echinococcus*. It is remarkable for its very small size—not much longer than an inch; and for the small number of its joints—which never amounted to more than three; and for the circumstance that the reproductive organs are confined to the two last segments, and the caudal joint separates as a *proglottis* at a very early period.

The last experiment to be noticed may be looked upon as the most interesting of all. Having the opportunity of repeating on a condemned criminal the experiments which he had previously performed on animals, Kuchenmeister contrived to give to this man, at seven successive times, between 130 and 12 hours previous to his execution, mingled with various articles of food, a number of *Cysticerci* from the hog and some from the rabbit. “They appear to have been partly disguised by their resemblance to the grains of rice in warm rice soup; partly by their likeness to the small bits of paste in a kind of vermicelli soup; and partly foisted on the unhappy wretch by being substituted for the small lumps of fat in blood-puddings” (*Brit. and For. Med-Chir. Review*, Jan., 1857, p. 119). After death, a number of young *Tæniæ*, in different stages of advancement, were found in the intestine; the greater number of them loose, but a few attached to the mucous membrane. The form of the hooklets, and other circumstances, induced him to regard these tape-worms as the *T. solium*. There were no traces of the *Cysticerci* last swallowed; and Kuchenmeister was of opinion that those only which were first taken, and which were quite fresh, had been converted into *Tæniæ*, and that those taken later, being dead, had been digested with the food. Leuckart made a similar experiment. He fed a man thirty years of age with *Cysticerci* from a pig, and in two months the man had *Tæniæ*.

But the enthusiastic Germans were not yet content with the proof. M. Humbert, of Geneva, experimented on himself. On the 11th of December, 1854, he swallowed fourteen fresh *Cysticerci* in presence of MM. Voget and Moulinié. Early in March of 1855 he felt the presence of *Tæniæ*, and discharged fragments of them.

With regard to the converse experiments, the following facts may be related:



sitic habitations. It appears by them to have been ascertained—(1.) That entozoa are always introduced into animals from without ; (2.) That some obtain access to the body of animals from water, or other matters in which they have previously lived in the free condition, while others are taken along with animal food in which the entozoa have lived parasitically ; (3.) That entozoa, when reaching sexual perfection in their parasitic condition, require to be in a situation which communicates with the external air, their most common position being the alimentary canal, and more rarely the pulmonary cavities ; (4.) That almost all the entozoa inhabiting close cavities, or encysted in the bodies of animals, are only imperfect and earlier forms of other entozoa, which may attain maturity in the open cavities of the same or of different animals, or in the free condition ; (5.) That entozoa rarely propagate themselves in the same animal in which they have arrived at sexual maturity, but require a different habitation, which they reach by migrations in the various modes before referred to ; (6.) That the cystic entozoa are the imperfect states of different *Tæniæ* ; (7.) That *Tæniæ* are almost invariably introduced, in their earlier condition, into the bodies of animals with flesh or other animal food ; (8.) That if the ova of *Tæniæ* be introduced into the alimentary canal of a suitable animal, through water, vegetable food, or fruit, their tendency is, after penetrating the tissues, to become encysted, and to assume the form of a cystic entozoön, such as that of a *Cysticercus*, *Cænurus*, or *Echinococcus* ; (9.) That if these cystic entozoa again are taken by certain animals with their animal food, the head part (which corresponds with that of a *Tæniæ*) resists digestion, and has a tendency to establish itself, and become developed into some form of *Tæniæ* in the alimentary canal, by the formation of segments after attachment to the mucous membrane.

Many of the immature entozoa pass their whole life as encysted parasites, and a few even acquire the jointed form, or become partially divided into segments, while still within their closed cysts. A well-known example of this is afforded by the *C. fasciolaris*, which inhabits cysts in the liver of the rat and mouse, and has been the means of leading Von Siebold and Dr. Henry Nelson (independently of each other's observations) to the discovery of the remarkable relation now proved to exist between the *cystic* or *vesicular entozoa* and the *cestoidea* or *tape-worms*. These observers found the cystic entozoa in the liver of the mouse and rat in every stage of development, from the simplest vesicular form of the true *Cysticercus* to that which (from the number of the joints and their external form) has all the appearance of a true tape-worm, and from which, in fact, it only differs in the absence of sexual organs within the segments. A careful comparison of the form of the head, its circle of hooklets, the four oscula or suckers, and other parts in the *Cysticercus* of the rat or mouse, with those of the *T. crassicollis*, which inhabits the intestine of the cat, has shown an exact resemblance between them. Dr. Allen Thomson has repeated and confirmed these observations (Art. "Ovum," *Cyclopædia of Anatomy*). The conclusion such observations lead to is now generally regarded as established—namely, that the



this *Tænia*, shall not furnish, by his excreta, the ova or embryos which, being taken accidentally with the pasture or water by the sheep, establish themselves in them as *encysted Cænuri*. Von Siebold states the important fact, that those flocks which are entirely without dogs, and are stall-fed, are never affected with the "sturdy."

**Prophylaxis**, therefore, is all-important; and the entrance of the *scolices* must be prevented. The following remarks are not less revolting than suggestive. Dr. Gordon, of the Army Medical Department, thus writes:

"*Tænia* appears to be of very frequent occurrence among the white troops in Upper India, and especially the Punjaub; and I have been told by some medical officers who have been stationed at Peshawur, our nearest cantonment to Affghanistan, that they firmly believe every third soldier has had tape-worm during the two years regiments remain there.

"From what I have been able to ascertain on the subject, natives are not particularly liable to tape-worm, and certainly not more so in the northwestern parts of India than in Lower Bengal. This is generally attributed to their almost total abstinence from animal food; and when we consider that both Hindoos and Mussulmans—all except the very lowest classes—abhor pigs' flesh, while our own countrymen are very partial to it, and the common soldier probably not very particular regarding the early history of the animal that is converted into pork for his use, an additional circumstance in favor of the transformation of the *Cysticercus* constituting the 'measles' of pork into *Tænia* is thus disclosed to us.

"Those who have escaped the misfortune of having had to pass some years in India can form no idea of the vast herds of lean, half-starved pigs that roam over the fields and waste grounds in the vicinity of villages; neither can they have any conception of the nature of the food on which these pigs subsist.

"The natives of India perform their ordinary natural functions in the open air on a piece of waste ground left for the purpose on the outskirts of every village, and where, morning and evening, men, women, children, and pigs dot the ground at short intervals from each other. In an incredibly short space of time after the villagers have left the field it is as clean as if they had never been there, while the herd by which the clearance has been effected may be found in some shady place near or close to a tank, with the exception of a few of the more insatiable, that have gone to hunt for dead dogs, cats, cattle, and Hindoos that have paid the debt of nature since the previous meeting, and have been thrown or left on the plain to be devoured by domestic animals or vultures.

"Pigs, however, are not the only animals that live in this filthy manner in India; cattle and sheep, that are so particular in their food in Britain, acquire degenerate tastes in India; and it is needless to enter into similar particulars regarding ducks, fowls, turkeys, and pigeons, all of which are more or less used as food by our countrymen there.

"I have thus alluded to these matters with a view to indicate some circumstances that most unquestionably tend to vitiate the quality of the animal food upon which our troops in India must subsist, and I think I have at least shown a sufficient cause for almost any amount of disease in the bodies of these animals; as also why their flesh should be more liable to become diseased in Upper India than in Lower Bengal" (*Med. Times*, No. 357, May, 1857).

Abstinence from the practice of eating raw meat is to be strenu-





fern (*Lastræa Filix-mas*) in doses of twenty to twenty-four grains—a remedy which by many is still believed to be the most efficacious. Dr. Gull's dose is one and a half to two drachms. The liquid extract of fern root is the officinal remedy of the British Pharmacopœia. It is made from the *rhizoma* or rootstalk of the *Aspidium* or *Nephrodium Filix-mas*, according to the formula: Fern root, in coarse powder, 1 part; ether, 2 parts, or a sufficiency: percolate and distil off the ether, and the liquid extract remains. The dose is  $\mathfrak{m}30$  to  $\mathfrak{m}60$ , in milk, or with mucilage, and should be given on an empty stomach. Mr. Squire finds the extract of the unexpanded frond equally effective with that of the rhizome. The powder may be used alone in doses of one to three drachms.

A remedy sometimes used in Germany is Chabert's *bandwurmöl*, or the "*oleum Chaberti contra Tœniam*." It is obtained by the distillation of twelve ounces of oil of turpentine mixed with four ounces of the *oleum animale fœtidum*, which is the crude oil obtained from hartshorn and animal bones.

A remedy of Abyssinian origin, called *Kousso* or *Cusso*, the flower of the *Brayera anthelmintica*, has recently been much recommended; and has doubtless been of great efficacy in some instances. It is administered in the form of powder, of which half an ounce is mixed with half a pint of warm water, and the infusion, *with the sediment*, is to be taken at two or three draughts, in the morning, on an empty stomach. If the bowels fail to be moved, a brisk cathartic ought to be given in three or four hours.

Another remedy, of more recent recommendation, is *Kameela* or *Reroo*, the *Rottlera tinctoria*, of the natural order *Euphorbiaceæ*, and sub-order *Crotonææ*. It has been highly lauded by Dr. Gordon, Surgeon to the 10th Regiment of Foot. He writes,—

"The success and rapidity of effect of the kameela in removing tape-worm in the cases of soldiers of the 10th Regiment, to whom I administered it, were such that I did not consider it worth my while to keep notes of them after the first two or three; nor, indeed, were the men to whom it was administered latterly taken into hospital, for they soon became aware of the wonderful efficacy of the remedy, asking of their own accord for a dose of it, after which they invariably parted with the worm in the course of a few hours, and then went on with their military duty as if nothing had happened; while, as I afterwards ascertained, considerable numbers did not think of 'troubling the doctor at all,' but, on suffering from the characteristic symptoms of the worm, applied for the kameela to the apothecary, and always with the same effect.

"We prepare a spirituous tincture by adding  $\mathcal{Oj}$  of alcohol to  $\mathfrak{z}iv$  of the powder, and then filtering. We never succeeded in obtaining more than  $\mathfrak{z}vj$  in this way; and of this  $\mathfrak{z}j$  in a little mint-water was generally found to be a sufficient dose,  $\mathfrak{z}ij$  being in some cases required, and perhaps in one or two,  $\mathfrak{z}iij$ , but I have never seen the remedy fail in removing the worm in a case where there were unequivocal symptoms of its presence.

"With kameela there is no unpleasant effect. It is not even necessary to take a dose of purging medicine as a preparative; and beyond a trifling amount of nausea and griping in some instances, no unpleasant effects are experienced; while by far the greater number of persons to whom it



Self-impregnation and copulation are both possible in these parasites. It is only very recently that the facts connected with the natural history of these parasites have been ascertained, and their general result may be stated as follows: The fully grown and sexually mature *Trematodes* (as exemplified in the *Distomata*) are parasites of the higher *Vertebrata*, and are oviparous.

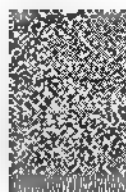
When the egg of the *Distoma* opens (by the springing open of a sort of hood, which gives it the operculated appearance at one end), it gives vent to an embryo which moves rapidly by means of cilia, as is the case with many infusoria, and especially of the *Opalina*, now regarded by Schultze, Agassiz, and Stein, as the earliest embryonic form of a *Distoma*. The discovery was made by Agassiz that a genuine *Opalina* (Fig. 18, *b*) was hatched from the egg (Fig. 18, *a*) of the *Distoma*. As such, they are found in sewage water (See A.

H. Hassall's *Reports on the Water of London*); also in the faeces of animals infested with liver flukes, their ova passing out with the bile. They die in pure water, as many vermicular animals do who would be more fortunate in water which is dirty, full of organic impurities, and abounding in food fitted for them.

From each of these ova is formed an embryo, in which no resemblance to the parent *Trematode* is to be recognized; but presenting the simple structure of a ciliated animalcule, like a polygastric infusorian known as the *Opalina*. This embryo is not itself converted by any direct process of development or metamorphosis into a perfect *Distoma*, but has a progeny gradually formed from germ-cells within it, and consisting sometimes of one, but more frequently of a number of bodies which, when they arrive at maturity, present each one an external form and internal structure and locomotive powers entitling them to be considered as independent animals. Nor are these directly converted into *Distomata*; a new progeny of animals is formed as before, nearly similar to those producing them, and equally differing from the complete *Distomata*. Each individual of this new progeny, as it increases in size, has formed within it, by development from germ-cells, the third progeny of the series and last of the cycle. These are different from their immediate parents, and in their internal organization soon manifest the type of the true *Trematoda*. They are endowed for a time with very active locomotive powers, to which a long caudal appendage contributes. Their progenitors have been confined in the parasitic condition, but these are in general freed from confinement, and move about with great vivacity for a time in the water surrounding the animals which their progenitors infested.

In this state they have been long known as *Cercariae*, having the appearance of minute worms with tails, and were classed by

FIG. 18.

Egg of *Distoma*.*Opalina*.



all obvious modes in which the *Cercariæ* of the *Trematoda* may find an entrance into the bodies of man and other animals.

Nine species of the fluke-like parasites have been found in man. They have been named as follows:

1. *Fusciola hepatica*, or *Distoma hepaticum*, in its full-grown condition, measures from eight to fourteen lines in length, and from three to six lines in breadth.

2. *Distoma crassum*.—In 1843 Mr. Busk found fourteen of these *Distoma* in the duodenum of a Lascar who died on board the "Dreadnought" hospital ship in the Thames. They are thicker and larger than those of the sheep, varying from one and a half to nearly three inches in length.

3. Of the *Distoma lanceolatum* only two instances are known of its occurrence in man.

4. Of *Distoma ophthalmobium* it is recorded that four specimens have been found in the eye of a child five months old, born with lenticular cataract. No one of them exceeded half a line in length; and they were situated between the lens and its capsule, where they could be recognized as so many dark spots on the surface of the lens (COBBOLD).

5. The *Distoma heterophyes* was found by Dr. Bilharz, of Cairo, in 1851, in the small intestine of a boy; and on a second occasion he collected several hundred specimens under very similar circumstances. The parts infested displayed a multitude of reddish points, due to the presence of dark-colored ova in the interior of the worms. The length of the largest specimens did not exceed three-fourths of a line (SIEBOLD, COBBOLD).

6. *Bilharzia hæmatobia* is so named by Dr. Cobbold in honor of its distinguished discoverer. It is, however, a bisexual parasite. The body of the male is thread-shaped, round, white, and flattened anteriorly. The oral sucker is triangular; the abdominal sucker at the end of the trunk is circular. Below this, at the curved margin of the abdomen, a furrowed canal exists for the reception of the female. This canal is peculiar and distinctive, and renders this *Distoma* generically distinct from the *Distomata* already noticed. The genital pore lies beyond the abdominal sucker and the commencement of the *canalis gynæcophorus*. The female is very thin and delicate; its tail is not provided with any canal. The suckers resemble those in the male; but the genital pore and the abdominal sucker are in contact. The length of the animal amounts to three or four lines, and the male is broader than the female (FRERICHS, MURCHISON, COBBOLD). Another name has therefore been given to it—namely, the *Gynæcophorus hæmatobius* (DIESING); but by whichever name it is known, it is of remarkable interest, not only from its peculiar anatomical structure, but from its great prevalence on the borders of the Nile, and from the grave and characteristic symptoms and appearances to which it gives rise. According to Griesinger it is met with in Egypt 177 times in 363 necropsies—i. e., equal to 33 per cent.

The first specimens were discovered by Bilharz, of Cairo, in the portal vein and its branches, and likewise in the walls of the urinary



These are all the symptoms which appear in connection with the urinary apparatus; and numbers of people of both sexes are affected in precisely the same way in certain parts of the Cape—as endemic hæmaturia—especially at Uitenhage and Port Elizabeth. In various samples of urine sent to him by a person suffering from this affection, he invariably detected the ova of the entozoön. Of these he was kind enough to give me specimens, and a copy of the wood-cut (Fig. 19 A). He was successful in observing the perfect ciliated embryo after its escape from the shell.

Dr. Cobbold has discovered the same *Distoma* in the portal system of an African monkey.

7. The *Tetrastoma renale*, as its name implies, infests the tubes of the kidney, and was discovered in 1888 by Lucarelli and Della Chiaje. It attains a length of five lines, has an oval, flattened body, and is furnished with four suckers disposed in a quadrate manner at the caudal extremity. The reproductive organs are situated near the mouth.

8. *Hexathyridium pinguiicola* was once found in a diseased ovary. The parasite attains a length of eight lines (TREUTLER, OWEN).

9. *Hexathyridium venarum* has been found in venous blood, and from the sputa of persons suffering from hæmoptysis (TREUTLER, CHIAJE, FOLLINA). It attains a length of three lines, is cylindricolanceolate, with six suckers biserially disposed on the under side of the so-called head (COBBOLD).

**Symptoms.**—The symptoms to which *Distomata* give rise in the human subject must of course vary with the site of the parasite; but nothing definite is known regarding them, except in the cases of hæmaturia and dysentery, already noticed. In sheep their presence occasions dilatation and catarrh of the biliary passages, accompanied by atrophy of the hepatic tissue. Jaundice rarely shows itself, and then only lasts a short time; but ultimately a condition of anæmia is developed, under which numbers of sheep die. The disease is known as "the rot" among sheep; and it prevails to a considerable extent among flocks feeding on marshy and wet land near the shores of rivers.

In the human liver bodies have been found like the ova of entozoa, so frequently met with in the liver of rabbits (GUBLER, quoted by FRERICHs).

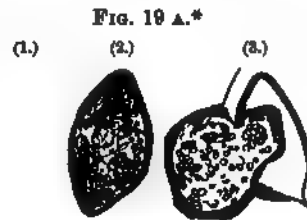


FIG. 19 A.\*

\* (1.) Ovum of *Distoma hæmatobium* from hæmaturia of the Cape of Good Hope (Dr. JOHN HABLEY); (2.) Embryo (ciliated) from ovum capsule; (3.) Embryo attached to the ovum capsule.





ago (1760–61) a student of Gottingen was dissecting the valve of the colon of a girl five years of age. He accidentally opened the gut, and several of these entozoa came out. Wrisburg and other students considered the worm a new one; but the demonstrator of anatomy maintained that it was an *Ascaris* or an *Oxyuris*, and a dispute arose. At last the new parasite got a name as a new worm, and was called a *Trichinalis* or *hairtail*. But it afterwards turned out that its *head* was hair-like, and not its *tail*, so it has been since called *Trichocephalus*. About this same time an epidemic raged in the French army stationed at Gottingen, and was described under the name of the *Morbus mucosus*; and this entozoön was frequently found in the bodies of the soldiers who died of this disease.

It is said to be very common in persons attacked with typhus fever; and is found in those dying with excessive discharges from the bowels, as in cholera and diarrhœa.

It is found in France, England, Egypt, Ethiopia, and rarely in Italy; abounding particularly in the caput cœcum. It is generally thought to be scarce in England—a persuasion which Dr. Cobbold thinks has probably arisen from “the negligence of pathologists, whose arduous duties connected with the superintendence of post-mortem examinations have, perhaps, left them little time for these inquiries.” On the other side of the Channel this parasite is so abundant in some localities that not less than one-half the inhabitants of Paris are affected by it (DUVAINE).

These parasites are males and females, in separate sexes, varying from  $1\frac{1}{2}$  to 2 inches.

The anterior extremity, carrying the head, is the narrow hair end, and it is usually buried in the mucous membrane of the intestines, while the remainder of the body moves freely in the midst of the mucous secretion, generally coiled upon itself.

The males are shorter than the females, and less thick posteriorly, with a long spiculum. The eggs are oval, with resisting shells  $\frac{1}{10}$ th of an inch in length.

**Generation of Round Worms.**—The generative organs of these nematoid worms are adapted for the reproduction of an enormous number of fertile ova. They are male and female; but the males, as a rule, are scarcer and smaller than the females.

The fertility of these animals is enormous. Dr. Eschricht has made an elaborate calculation regarding the *A. lumbricoides*, the commonest parasite of man. The ova being arranged like flowers upon a stem in the ovary tubes, he has counted fifty in a circle, or in every transverse section. The thickness of each ovum he estimates  $\frac{1}{800}$ th of a line ( $= \frac{1}{800}$ th of  $\frac{1}{2}$ th  $= \frac{1}{1600}$ th of an inch); so that in every line of length of the worm there would be 500 wreaths of 50 eggs each, = 25,000 eggs.

The length of each horn of the uterus is taken at sixteen feet, which gives 2304 lines; and for the two horns it will give 4608 lines. The eggs, however, gradually get as large as  $\frac{1}{80}$ th of a line, so that only sixty wreaths of eggs come to be on one line, or about 3000 ova; and an average gives 14,000 ova in a line—i. e., *sixty-four millions of ova in every mature female Ascaris*.



bibed the fluid, while the remaining part continued shrivelled up, and adherent immovably to the board. I have seen the very same results in the *Ascaris* which infests the peritoneal covering of the mackerel. Such being the tenacity of life on the part of the mature animal, how much more do the ova possess the powers of endurance? Without losing latent life, they even develop themselves under circumstances of the most improbable kind. Dr. Henry Nelson and Dr. Allen Thomson have observed the development of the ova of the *A. mystax* to proceed for several days, while the parent bodies containing them were immersed in oil of turpentine. I have once seen the same occurrence; and also I have seen the development of the embryo proceed in spirits of wine for about three weeks before signs of vitality had ceased.

**Periods of Incubation of the Ova.**—The eggs are ovoid, and covered by a transparent envelope or chorion, which after fecundation and segmentation becomes tuberculated. Hence the various accounts given as to their surface appearance. They are expelled with the fæces in the case of the *A. lumbricoides*. They have been placed in water and taken care of for various lengths of time, and Richter records that at the end of eleven months each ovum contained a living embryo. In August, 1853, Verloren and Richter put a fragment of a mature female *Ascaris* (*marginata* of the dog) into water, so as to keep the ova moist merely; and he examined them from time to time with the microscope. Segmentation having commenced, the development of the young was completed in fourteen days. They moved with great briskness within the egg-shells, *but did not break through them*. In this imprisoned or encysted state they continued throughout *autumn* and *winter*; the movements of the embryos gradually diminishing and at last entirely ceasing during the winter months, to recommence in the following spring, and become again distinct in summer; *but they never broke through the shell*.

The condition of these *Ascarides* from the encysted state of inclosure within the egg is only changed under favorable circumstances—namely, when the animals are liberated and carried on to further development; and now it is known that the embryo of nematoid worms may pass the winter in a torpid state, floating about in the open waters, or lying in moist places. The fully formed embryo is cylindrical, its length  $\frac{1}{10}$ th of an inch; the mouth is not furnished with the three characteristic papillæ of the genus, and the tail terminates suddenly in a point. It is highly probable, from the evidence, that the embryos are directly transferred to the alimentary canal of man from river- and pond-water.

4. The *Oxyurus vermicularis* was known to Hippocrates, and is one of the most troublesome parasites of children, and occasionally of adults. It is a minute, white, thread-like worm, the male being about a line and a half in length, and the female five or six lines. They inhabit chiefly the rectum, where they are often found in clusters, rolled up in balls of considerable size, and from the rectum may creep into the vagina or urethral orifice. Sometimes they give rise to profuse and exhausting bloody discharges from the vagina.



nematode helminthe; the male in its fully developed and sexually mature condition, measuring only  $\frac{1}{8}$ th of an inch, whilst the perfectly developed female reaches a length of about  $\frac{1}{4}$ " ; body rounded and filiform ; usually slightly bent upon itself, rather thicker behind than in front, especially in the males ; head narrow, finely pointed, unarmed, with a simple central minute oral aperture ; posterior extremity of the male furnished with a bilobed caudal appendage, the cloacal or anal aperture being situated between these divergent appendages ; penis consisting of a single spicula, cleft above, so as to assume a V-shaped outline ; female shorter than the male, bluntly rounded posteriorly, with the genital outlet placed far forward, at about the end of the first fifth of the long diameter of the body ; eggs measuring  $\frac{1}{1270}$ " from pole to pole ; mode of reproduction viviparous. (2.) The sexually mature trichina inhabits the intestinal canal of numerous warm-blooded animals, especially mammalia (also of man), and constantly in great numbers. (3.) At the second day after their introduction, the intestinal trichinæ attain their full sexual maturity, lose their spiral figure and become stretched, whilst they grow rapidly, and their generative organs are developed. (4.) Most females contain from three to five hundred ova. In six days the female parasites will contain perfectly developed and free embryos in the interior, and these on attaining full size pass out at the vaginal opening. The eggs of the female trichina are developed within the uterus of the mother, into minute filaria-like embryos, which from their sixth day are born without their egg-shells. (5.) The new-born young soon afterwards commence their wandering. They penetrate the walls of the intestines, and pass directly through the abdominal cavity into the muscles of their bearers, where, if the conditions are otherwise favorable, they are developed into the form hitherto known. (6.) The direction in which they proceed is in the course of the intermuscular connective tissue. (7.) The majority of the wandering embryos remain in those sheathed muscular groups which are nearest to the cavity of the body (abdomen and thorax), especially in those which are smaller, and most supplied with connective tissue. (8.) The embryos penetrate into the interior of the separate muscular bundles, and here already after fourteen days, acquire the size and organization of the well-known *trichina spiralis*. (9.) Soon after the intrusion of the parasite, the infested muscular fibre loses its original structure. The fibrillæ collapse into a finely granular substance, whilst the muscular corpuscles change into oval nucleated cells. (10.) The infested muscular bundle retains its original sheathing up to the time of the complete development of the young trichinæ, but afterwards its sarcolemma thickens and begins to shrivel at the extremities. (11.) The spot inhabited by the rolled-up parasites is converted into a spindle-shaped widening, and within this space, under the thickened sarcolemma, the formation of the well-known lemon-shaped or globular cysts commences by a peripheric hardening and calcification. One cyst may have from one to three trichinæ. (12.) The migration and development of the embryos also take place after the transportation of impregnated trichinæ into the intestines of a new host. (13.) The further development of the muscle trichinæ into sexually mature animals is altogether independent of the formation of the calcareous shell, and occurs as soon as the former have reached their completion. The male and female individuals are already recognizable in their larval state (LEUCKART, COBBOLD\*.)]

---

\* [*Entozoa: an Introduction to the Study of Helminthology, with reference more particularly to the Internal Parasites of Man.* By I. Spencer Cobbold, M.D., F.R.S. London, 1864.]

The non-encysted *Trichina* may exist in the flesh of animals without being visible to the naked eye. In the encysted state they are difficult of detection without the aid of a lens, if cretification has not commenced in the cyst. The cysts are round or elongated, and appear, according to their shape, like small round dots, granules,

FIG. 20.\*

FIG. 21.†



vesiculæ, or streaks, grayish-white or opaque, and quite distinct from the red transparent muscle. When the cyst has become calcareous, its limy material may be dissolved away by acetic or hydrochloric acid with the evolution of gas, and the parasite is then seen coiled up within (Fig. 20).

The symptoms of the disease induced by this parasite are at first of a febrile nature, having a close resemblance to some forms of specific fevers. Dr. Philip Frank, lately Assistant-Surgeon on the staff of Her Majesty's Army Medical Department, was the first to send an account of this remarkable disease from Germany to this country. He described a case of its occurrence in the *Medical Times and Gazette* of May 26, 1860; and recently Dr. Parkes has given a short notice of *trichina disease* in the *Sanitary Report of the Army Medical Department* for 1860, p. 351. The history of the case referred to by Dr. Frank is as follows:

In January, 1860, a servant girl about twenty years of age died in the Dresden Hospital from the effects of the *T. spiralis*; and the muscles of her body furnished materials for numerous observations and experiments, which have thrown much light on the origin and development of this parasite. The illness of the patient commenced about Christmas, 1859; and the symptoms may be arranged into two sets,—(1.) Extreme lassitude, depression, sleeplessness, loss of appetite, and eventually febrile phenomena, which were well expressed, so much so, that the case was set down as one of typhoid fever; but grave doubts prevailed, for (2.) A new train of symptoms developed

\* Slightly magnified cyst of *Trichina spiralis* (after VISCNOW).

† The *Trichina spiralis* removed from its cyst (after VISCNOW).

themselves—namely, the whole muscular system became the seat of excruciating pain, especially in the extremities. Contractions of the knee and elbow joints supervened, and associated with such extreme pain as to render extension of the limbs impossible. Edema of the legs followed; and the case terminated fatally by pneumonia, about the twenty-eighth or thirtieth day after the first symptoms of illness.

A post-mortem examination of the body showed the muscles moderately developed, of a pale reddish-gray color, and dotted over with specks, which turned out to be groups of non-encapsuled *Trichinæ*, lying free upon and within the sheaths of the muscular fibres. They were alive—some coiled up and others lying straight; and they appeared to be in all stages of development, diffused throughout all the striated muscles of the body, not even excepting the heart itself. They abounded in such vast numbers that as many as twenty *Trichinæ* were seen in the field of view through a low magnifying power, the muscular tissue being everywhere in a degree of very marked degeneration. In the jejunum were found sexually mature *Trichinæ*. Death was due to the development of the *T. spiralis*, whose existence fully explained the anomalous symptoms which attended the case.

On looking into the history of the girl it was found out that she had been a servant in a farm-house, and had been taken ill very soon after the killing of two pigs and an ox—animals which it is customary to kill about Christmas.

Pigs are known to be infested with the *T. spiralis*—so are oxen; and Professor Zenker went to the master's house, and found some ham left of the identical pig that had been there killed, and also some sausages. The flesh of the pig was examined microscopically, and every specimen examined showed that the pig's flesh was infested with *Trichinæ* in the encysted state. At the same time Professor Zenker learned that, soon after the girl had been taken ill, the housekeeper became unwell, with similar symptoms, but in a less severe degree; and all the servants about the farm became more or less ill about the same time. The house of the butcher who had killed the pig was then visited by Professor Zenker, who was informed by the wife of the butcher that he had been very ill since that event. He had been three weeks in bed, suffering from *rheumatic pains in the limbs*, and had been as if paralyzed over his body—unable to move his arms, legs, or neck. He had never suffered anything of the kind before, but had always been a healthy and strong man. He thought he had caught cold the day he killed the pig; but when it is known to be a habit of German butchers to taste the meat they kill, in the raw condition, the history of these cases, to Professor Zenker, became a history of *trichinatus disease*—the development and growth to maturity of the *T. spiralis* in the muscles of those who lived at the farm-house, as well as of the butcher who had killed the pig, and who no doubt had eaten some of its flesh.

Numerous experiments were made with the flesh of the girl who died in this remarkably morbid state. Portions of the flesh were



sent by Zenker to Professor Virchow at Berlin. He fed a rabbit with some of it, and this rabbit died about a month after the feeding, with symptoms of general muscular paralysis, and myriads of young *Trichinæ* were seen in its muscles. Other rabbits were fed with the flesh of the first rabbit, and they too died with similar phenomena.

Another observer had before made similar experiments. Herbst, in 1852, fed three young dogs with the flesh of a badger whose muscles were saturated with *Trichinæ*. The dogs in their turn became *trichinatus*; being killed after a few months, the parasites were seen in their flesh. Pigeons also were fed with moles' flesh known to be *trichinatus*; and free *Trichinæ* were found in the flesh of the neck, the wings, and the thighs of the pigeons in eighteen days. But Herbst did not examine into the relation between the capsuled and the free *Trichinæ*, as Virchow and Zenker have done.

The *Trichina spiralis* is now well known not to be limited to the muscles of man. It occurs in eels, cats, dogs, badgers, hedge-hogs, pigeons, moles, and swine.

Virchow found the villi of the intestines of the rabbits loaded with the ova or *prospermiæ* of the entozoa; and he found mature *Trichinæ* of both sexes moving freely in the mucus of the intestine. The males were filled with sperm corpuscles, while the females were densely stocked with ova and their germs, and with young ones in the eggs, coiled up like little snakes.

[It has been found also in the horse, ox, sheep, and other ruminants, also in rabbits, rats, mice, and guinea-pigs, which have been fed with trichinous flesh. The debris of an animal devoured by carnivora may become fatal to rodents, or a carcass near a marsh or rivulet may communicate the parasites to the ruminants which drink the water, or to pigs (DAVAINE).]

In the summer of 1860 a subject was received into the dissecting-room of the University of Edinburgh; and the muscles of that subject contained numerous flesh-worms. Dr. Turner, the demonstrator of anatomy, then took the opportunity of verifying the experiments of the German professors. He fed kittens with portions of the human flesh containing the worms, which were observed to move, though somewhat languidly, on rupturing the cysts. To one cat on the 7th, 13th, and 16th of July he gave portions of the flesh, and in the intervals fed it on bread, milk, and fish. He killed the cat on the 24th of the month. Nothing could be seen with the naked eye in the fluid of the small intestines; but on placing a drop below the microscope, thread-like worms were seen actively moving about in it, or coiling themselves up in a spiral form. Every drop of fluid taken contained one or more. Each of the thread-like worms was about  $\frac{1}{40}$ th of an inch long and  $\frac{1}{1000}$ th of an inch broad, with a pointed and rounded end, and about two thirds smaller than the mature flesh-worms met with in the muscles of the cat. These had migrated from the intestines, and after working their way between the fibres of the muscles, had become encapsuled—the cap-

sules being perfectly transparent. Herbst and Virchow have found the flesh-worms both in the mesenteric glands and in the mesentery, and therefore, presumably, *in transitu* between the intestines and the muscles. All the phenomena described occurred within the space of a single month; and even as early as three weeks after feeding, Virchow found the young brood equal in size to those administered at the commencement. The genesis, development, and migrations of these flesh-worms are thus proven to be astonishingly rapid. Dr. Thudichum has also very recently verified these experiments; and at the conversazione of the British Medical Association, held at Downing College, Cambridge, on 4th August, 1864, he exhibited the parasite, *living*, in various stages of development, which he obtained from the muscle of a rabbit infested with them, and also from some pork chops (*Brit. Med. Journal*, August 13, 1864).

Thus the *T. spiralis* has been shown to be a bisexual parasite, producing its young alive in the intestines of the animal whose muscles it may infest, either in the free or in the encapsuled state. So long as it remains in the capsule it is immature and non-sexual, and so far they are harmless; but should they become free, they grow larger, and may become mature, and so give rise to others, if the death of the animal they inhabit does not occur.

In the mucus of the intestines the mature *Trichinæ* find a suitable place for growth and breeding, their progeny finding their way to the muscles, where they eventually become encysted; and their favorite haunt there seems to be the small muscles of the larynx (ZENKER).

According to Virchow's conclusive testimony, all these phenomena occur within the space of a single month; and, in his experiments, even as early as three weeks after ingestion, the young were found to equal in size those that he administered at the commencement. The genesis and migrations of *Trichinæ* are therefore astonishingly rapid, and probably without parallel in this class of parasites (COBBOLD).

Since the discovery by Leuckart of the round worm, of which the *T. spiralis* is the immature condition, since the case recorded by Zenker, and since the more complete knowledge that has been acquired by experiments, of the wonderful migrations of the young *Trichinæ*, attention has been especially directed to the possibility of the *trichinatus* disease in man being more common than was anticipated.

In December, 1860, Professor Wunderlich met with a case of prolonged fever, which did not correspond in its course with any of the well-known specific fevers. The patient was a butcher. He eventually got quite well, and so far negatived the diagnosis of *acute* tuberculosis which had been made. A second butcher, from the same establishment, came into the hospital with the same symptoms of high fever, with immense depression; but the course of the disease again did not correspond with any of the known fevers. Here, as in the first case, the muscles were particularly implicated, but in a less degree. There was not only muscular pains, but absolute soreness of the muscles on pressure. This man, too,

eventually got well. A third and a fourth butcher, from the same house; were also taken ill with similar severe febrile symptoms, but they were not seen by Wunderlich.

These men had been killing a number of pigs; and, as is the custom, they ate of the raw flesh. Eight men so ate, and four of them were afterwards attacked with these anomalous but severe febrile symptoms. Unfortunately, none of the pork had been preserved, and the possibility of *Trichinæ* existing in it had therefore not been proven. Moreover, none of these men died, and no evidence of the parasite existing in their muscles was obtainable. But looking to the undoubted fact that the use of the raw meat brought on the disease, and to the great probability that the wanderings in large numbers of the *Trichinæ* will produce these symptoms, Professor Wunderlich deems himself justified in thinking that there are some grounds for considering these febrile attacks to have been due to *trichinatus* disease. That individuals enjoy good health although the muscles are infested with the encapsuled *Trichinæ* is now well known from the number of cases that have been seen in dissecting-rooms. Cases are also referred to by Mr. Curling, of its being recognized in the muscles of men killed by accident, when engaged in severe manual labor (*London Med. Gazette*, Jan., 1838; also Turner in *Edin. Med. and Surg. Journal*, 1860, p. 209). The distinguished teacher of clinical surgery at Berlin, Professor Langenbeck, related to the Medical Society there, in 1863, the case of a man from whom he had recently removed an epithelial cancer situated in the neck. During the operation the *platysma myoides* exhibited a singular appearance, which, on careful inspection, was found to arise from the presence in the muscle of innumerable dead *Trichinæ* contained in calcified capsules. On inquiry, the following facts were elicited: In the year 1845 there was a "church visitation" (whatever that may mean), in which eight persons took part, and of these, seven afterwards sat down to a breakfast consisting of ham, sausages, cheese, roast veal, and white wine. In the course of three or four days every one of the seven persons was seized with diarrhœa, pains in the neck, œdema of the face and extremities. Of the seven, four died, and the three who survived (among whom was the man operated upon eighteen years afterwards by Professor Langenbeck) remained ill for long afterwards. The suspicion arose that poisoning, through the agency of white wine, had taken place; and an investigation was made, but without any result. The innkeeper, however, at whose house the breakfast was given, being still under suspicion, was obliged to give up his business and emigrate. The importance of such a case in all its forensic aspects cannot be over-rated; and it becomes an important subject of inquiry whether some of our cases of death from suspected but unproved poisoning may not be due to *trichina* disease, which is now known to be much more prevalent than has hitherto been supposed, both in this country and in Germany.

Very recently attention has been again awakened on the subject by an occurrence almost tragical.

About the middle of October, 1863, there was a festive celebration at Heltstädt, a small country town in Prussia, near the Hartz Mountains, numbering from 5000 to 6000 inhabitants. One hundred and three persons sat down to an apparently excellent dinner, mostly men in the prime of life. Within a month more than 20 persons had died, and more than 80 persons were then suffering from the fearful malady, while those who were apparently unscathed were in hourly fear of an outbreak of the encapsuled flesh-worms.\*

The dinner had been ordered at a hotel, and it was arranged that the introduction to the third course should consist of "Röste-wurst." The sausage-meat was therefore ordered at the butcher's the necessary number of days beforehand, in order to allow of its being properly smoked. The butcher, on his part, went to a neighboring proprietor of pigs, and bought one of two pigs from the steward of the pig-farm. The steward unfortunately sold a pig which his master intended should not be sold, because it was not considered to be in good condition. Nevertheless, for this time at least, the butcher got "the wrong sow by the ear." The ill-conditioned pig was the one that was killed and worked up into sausages. These were duly smoked and delivered at the hotel; and after being toasted before the fire (so as to be warmed through merely) they were served to the guests at the dinner-table.

On the day after, several persons who had eaten the dinner were attacked with great irritation of the bowels, loss of appetite, great prostration, and fever. The number of persons attacked rapidly increased; so much so, that great alarm was felt in so small a town lest an epidemic of typhoid fever was about to set in.

But one of the physicians at last conjectured that some poison must be at the bottom of the outbreak, and an active inquiry into all the circumstances of the dinner was instituted; and when the muscles of the calves of the legs of some of the sufferers began to be affected, the descriptions of Zenker's case (already described) was at once remembered. The remnants of sausages, and of pork employed in the manufacture of them, were examined with the microscope, and found to be literally swarming with encapsuled flesh-worms. From the muscles of several of the suffering victims small pieces were excised, and under the microscope they were seen to be charged with *Trichinæ* in all stages of development. It could, therefore, no longer be doubted that as many of the 103 persons as had dined together and partaken of the "*Röstewurst*" were affected with trichinous disease by eating the trichinous pork, the flesh-worms of which had not been killed by the smoking and toasting. On the contrary, the subdued heat of toasting would rather foster their vitality.

This catastrophe awakened sympathy and fear throughout the whole of Germany. Most of the leading physicians were consulted in the interest of the sufferers; and some visited the neighborhood

---

\* [See the report of this outbreak of Trichiniasis, by Dr. Thudichum, in the Seventh Report of the Medical Officer of the Privy Council, London, 1864.]

where most of the affected patients were. None could bring relief or cure. Case after case died a slow and lingering death, by exhaustion from nervous irritation, fever, loss of muscular power, inflammation of the lungs, or of organs essential to life. The cases have been observed with great care and chronicled with skill. All the features of the remarkable disease have been registered in such a manner that hereafter there can be no difficulty in recognizing the disorder.

The disease begins a few days after eating the meat in which there were *Trichinæ*, with loss of appetite, and, almost without exception, with diarrhœa and fever; œdema of the eyelids; also pain, or at least painful sensation of weakness, in the limbs; œdema of the joints; difficulty in moving the tongue; profuse clammy perspiration; and those patients who do not become convalescent die either unconscious, with symptoms of typhoid fever, or, in a few cases, remain conscious to the end, complaining of inability to breathe freely.

[There would appear to be three stages in the disorder: (1.) Until the progeny are born; lasts from four to eight days; the symptoms, general discomfort, gastric trouble, especially after eating, vomiting, and diarrhœa. (2.) From the beginning of their wanderings, till they are encysted in their permanent resting-places; the symptoms, fever, and profuse perspirations; œdema of the face, sometimes extending to the whole body, inflamed conjunctivæ, photophobia, and pain on moving the eyes, especially on looking upward; scanty, high-colored urine, diarrhœa and tenderness of the abdomen; rigidity, swelling, weariness, formication, and severe pain on moving the muscles, first, of the neck and back, next of the arms and thighs, then of the fore-arms and legs; breathlessness and hiccough come on when the respiratory muscles are infested, hoarseness and loss of voice on invasion of the laryngeal muscles, and difficulty of mastication and deglutition on the muscles of these functions being affected. The sufferer lies on his back, with his legs drawn up, unable to move or speak. The fever continues or increases; typhoid symptoms set in; there are much exhaustion, meteorism, restlessness, and sometimes delirium; great wakefulness appears, often very early, and fainting fits. Lobular pneumonia may intercur, with pleural effusion. This stage may end in death within four to six days, though commonly it lasts three to five weeks. (3.) The symptoms lessen on the reappearance of the urinary secretion, but stiffness of the muscles may last for some time, and baldness and epidermic desquamation may take place. In milder cases, there is a feeling of general discomfort, restlessness, lassitude, vertigo, loss of appetite, thirst, wakefulness, or disturbed sleep, lumbar pain, swollen face, and diminished urine. Its attacks are said sometimes to be very insidious, and the patient who has not been severely ill, dies suddenly from pneumonia or peritonitis. Children are said to suffer less than adults.]

The only important symptom of typhoid fever absent in the disease is the enlargement of the spleen; and it is very probable that some of the so-called epidemics of typhoid fever in former days were caused by the propagation of *Trichinæ* in the human body.

[There should be no difficulty in the differential diagnosis of the trichin-



ous disease and typhoid fever. There is nothing in common in the distinctive symptoms of the two disorders. The epistaxis, the characteristic physiognomy, the pain and gurgling on pressure in the right iliac region, the rose-colored eruption of typhoid fever, are all wanting in the trichinous disease; while in the latter, the early troubles of the digestive functions, followed by œdema of the face, and severe muscular pain, especially on motion, with the breathlessness which often worsens to threatened asphyxia, present a series of symptoms in regular and close sequence, corresponding to the successive epochs of the larval production in the intestines, and the migration to and abode in, the muscles, which happens in no other disorder.]

Since the disease has been known (about five years ago), a great many cases have been observed in Germany.

[In 1860, Dr. Zenker recognized the disease in Dresden, and on examining the ham and sausages that had been eaten by the persons affected, found them infested with trichinæ. In 1859, 1860, and particularly in 1862, many cases were noticed at Blankenbourg, chiefly amongst the soldiers. In 1862, of 60 attacked, 2 died (SCHOLZ). Two cases were seen by Wunderlich in Leipsic in 1861; and Wagner describes 5 cases which occurred there in 1863 (*Archiv der Heilkunde*, 1864). Landois met with 12 cases in the island of Rügen in 1861, and Wentzel with 20. In that year, at Cosbach, 3 persons of the same family, who had eaten of fresh pork, and in whose muscles Zenker found trichinæ, were affected. In the same year, 300 fell ill with the disorder in Magdebourg, and 2 died. In the summer of 1862, at Calbe, 30 persons, in a population of 1200, were attacked—9 males, 25 women, and 4 children, and 8 died—1 male, 6 women, and 1 child. In the spring of 1862 there was an outbreak at Plauen, in Saxony, and several died (BÖHLER). In the autumn of 1863 the Hettstädt epidemic occurred, already described. There was an outbreak at Hedersleben in 1865 (300 cases and 40 deaths); at Zittau, in 1866 (57 cases); and at Görlitz (80 cases and 1 death).

But few cases of the trichinous disease have been recognized in the United States. The first cases reported are believed to be those of Dr. Joseph Schnetter, of New York; 2 cases after eating underdone pork-steaks; neither were fatal (January, 1864); and 5 cases and 1 death (February, 1864), of persons who had eaten raw ham, in which trichinæ were subsequently found.\* About the same time, Dr. Voss, of New York, had 4 cases on board one of the Bremen steamers, then in the harbor. Dr. Voss verified his diagnosis by cutting down on the deltoid muscle of one of the affected persons, and removing a portion for microscopical examination; it proved to be filled with trichinæ.† Dr. J. R. Lothrop, of Buffalo, has reported a case.‡ Nine cases have happened in 1866 in one family at Marion, Iowa, and been reported by Dr. Joseph H. Wilson.§ About the 5th of May, six persons in the family of Mr. Bemiss, of that place, were taken ill, with the characteristic symptoms of the trichinous disease, which was not, however, at first recognized, and the disorder

---

\* [*Observations on Trichina Spiralis*. By John C. Dalton, M.D. The Transactions of the New York Academy of Medicine, vol. iii, 1864.]

† [Dalton, *l. c.*]

‡ [*A Treatise on the Principles and Practice of Medicine*. By Austin Flint, M.D., 1866.]

§ [*St. Louis Medical Reporter*, July 15, 1866; *Chicago Medical Journal*, August, 1866.]



town of Brunswick, North Germany, where a most careful inspection of 19,747 hogs was made in the years 1864-65, only two were found to have trichinæ in their muscles; "the proportion being 1.10000 to 1.50 in the Chicago pork."\* One of the tables of the Chicago committee shows the great variation in the number of helminthes infesting the several muscles examined. An approximation only to the number existing in a cubic inch of a given muscle could be obtained. The method adopted, was to count the trichinæ existing in several different portions of a muscle, each a cubic one-tenth of an inch in size, and to multiply the average number to a cubic inch. Of twenty-eight specimens examined with this view, only three of them contained over 10,000 to the cubic inch,—18,000, 16,000, and 15,000, respectively. The remaining twenty-five were infested to a much less degree—from 48 to 6000 in the cubic inch. It was calculated that a person eating an ordinary meal of the pork from which the specimen containing 18,000 to the cubic inch was taken, would soon become infested with not less than 1,000,000 of young trichinæ.†

With regard to the muscles of the hog which are the most common site of trichinæ, the observations of the Chicago committee do not agree with those of European observers. In Germany, the inspectors of pork are instructed to examine microscopically nine different sets of muscles, namely,—those of the diaphragm, tenderloin, shoulder, front and back of neck, extensors of the fore-arm, flexors of the leg, and the muscles of the larynx. In the trichinous-infested muscles examined by the Chicago committee, more than one-half were spinal muscles, which are not named in the German list.

In conducting an examination of the trichinatus pork, the tendinous extremities of muscles should be selected, as here they are usually most numerous. The cysts are visible to the naked eye as whitish, round, or ovoid specks, sprinkling the surface of the muscle. If a very small piece of muscle is cut off with scissors, and then torn in shreds with a needle, freeing the cysts from the flesh, and these are touched with a drop of hydrochloric acid, the lime is dissolved and the white coloring disappears; or a piece of the suspected flesh may be put into a watch glass with liquor potassæ (1 part to 8 of water), when it becomes changed to a mucus-like, clear mass, and the capsules will be seen as sharply defined minute white specks (LEUCKART). But it is always better, if possible, to use the microscope, and trichinæ not yet encysted can only be recognized by the microscope. A thin layer of the suspected flesh should be cut out with a sharp knife, and spread over a glass plate, moistened with a drop of water, covered with a thin piece of glass, and examined by a magnifying power of 50. Their intimate structure cannot be recognized with a less power than 200 (ALTHAUS).‡]

The vitality of the *Trichinæ* is not destroyed unless the meat or other substances in which they are located be subjected to the tem-

\* [Chicago Medical Examiner, April, 1866.]

† [As many as 2,000,000 trichinæ have been estimated in the muscles of a man who died of the disorder, and Prof. Dalton counted in a piece of muscle (in one of Dr. Schnetter's cases)  $\frac{1}{2}$ th of an inch square, and  $\frac{1}{30}$ th of an inch thick, where they were in average abundance, twelve trichinæ, which would give in round numbers, over 85,000 to the cubic inch; and in the portion of muscle taken from the living subject, in Dr. Voss's case, they numbered a little over 7000 to the cubic inch (Dalton, l. c.). In one of Dr. Wilson's cases, which proved fatal, 104 trichinæ were counted in a piece of the rectus femoris muscle measuring  $\frac{1}{2}$ th of an inch square, and  $\frac{1}{2}$ th of an inch thick, which would give nearly 180,000 to the cubic inch.—EDITOR.]

‡ [On Poisoning by Diseased Pork: being an Essay on Trichinosis or Flesh-worm Disease; its Prevention and Cure, by Julius Althaus, M.D. London, 1866 ]





[It would appear that emetics and active cathartics, if employed in the first stage of the disorder, while the parasites are yet chiefly in the alimentary canal, are of use. It is recommended that large doses of olive oil be subsequently given. The sleeplessness and copious perspirations are best relieved, according to Ruprecht, by wet packing. But some substance, which is capable of destroying the trichinæ, without damaging the bearer, is desirable. Dr. Mosler, from his experience with the disorder at Quedlingburg in 1863, is of opinion that the only rational treatment for the trichinous disease in man is benzine. His formula is: *Benzine*, fʒij; *liquorice-juice*, *mucilage of gum arabic*, āā fʒj; *peppermint water*, fʒiv. *A tablespoonful to be given every hour or two hours.* Dr. Mosler states that in this form benzine is well borne, and that none of his patients felt any bad effects from its use. It was largely used in the Hedersleben outbreak, but with no good results. Fiedler has shown that the pretended cures with *carbazotic (nitro-picric) acid*, reported by Friedreich, were mere coincidences, and he proposes *Dippel's animal oil*. Dr. Tavernier suggests *carbolic acid*. Küchenmeister speaks favorably, in recent cases, of equal parts of turpentine and sulphuric ether. At best they can only kill the trichinæ in the intestinal canal; when these begin to migrate, all remedial measures must be unavailing; and if recovery takes place, it is because the parasite is finally nested in its calciform cyst.]

6. The *Sclerostoma duodenale* is known to be tolerably common throughout Northern Italy; and, according to Pruner, Bilharz, and Griesinger, it is so remarkably abundant in Egypt that about one-fourth of the people are constantly suffering from a severe anæmic chlorosis, occasioned solely by the presence of this parasite in the small intestines.

"Its length is about one-third to half an inch, its width about one-twentieth of its length. Its head has a round apex, and its extremity, which is bevelled at the expense of its posterior surface, is provided with hooklets that occupy converging papillæ. The mouth contracts, to open into a thick muscular pharynx, which, widening as it passes downward, ends, after occupying one-seventh of the body, in the intestine. The sexual differences of the male and female are very interesting. Its pathological significance is chiefly due to the hemorrhage caused by these parasites, which are often present in thousands between the *valvulæ conniventes* of the duodenum, jejunum, and ileum, and not infrequently in the submucous areolar tissue. In short, the physician practising in Egypt must never forget that the chlorosis of this climate is often the result of repeated and small hemorrhages from the intestine caused by these parasites. Turpentine, as Griesinger points out, promises to be the best remedy both as a styptic and as a vermifuge" (*Brit. and For. Med.-Chir. Review*, l. c.).

7. The *Strongylus bronchialis* was first discovered by Treutler, in 1791, infesting the enlarged bronchial glands of an emaciated man. The parasite is cylindrical, slightly narrowed anteriorly, filiform, but somewhat compressed at the sides, semi-transparent posteriorly, and of a blackish-brown color. It measures from half an inch to three-quarters of an inch in length.

8. The *Eustrongylus gigas* is fortunately rare in man, though common in a great variety of animals, such as weasels. It inhabits

the kidney, destroying the substance of the organ, the walls of which become the seat of calcareous deposits.

9. The *Speroptera hominis* is furnished with a spiral tail, having peculiar marginal appendages.

10. The *Filaria medinensis*, Guinea worm, or *Dracunculus*, lives amongst the connective tissue of man and of some animals. In this situation it is only known as a female, containing an enormous quantity of young *Filaria*, and resembles a long piece of uniformly thick white whip-cord. In this country few are familiar with its appearance, or with the lesions it produces; and we therefore look for our knowledge regarding the main points in the natural history of this parasite to be furnished to us by the observer in Africa or Asia; and of whom we hope that they will fill up the gaps which still exist.

The Guinea worm is essentially a tropical parasite. It is endemic in the hot intertropical regions of Asia and Africa, extending from Egypt, about 23° or 24° north latitude, to Sumatra and adjacent islands, as far as 10° or 12° south. But it is only in some districts within these tropical limits that the parasite abounds. For example, it is endemic in Arabia Petrea, the borders of the Persian Gulf, and of the Caspian Sea, the banks of the Ganges, Upper Egypt, Abyssinia, and Guinea. Its occurrence in Guinea (although it has its common name from this place) is extremely capricious. In some districts every native who comes off to the ships seems to be affected by it; in other places in Guinea it is very rarely seen.

The *F. medinensis* is unknown in America, unless the person in whom it exists has been in the places where the *Dracunculus* is endemic. The only exception is the island of Curaçuo. It is sometimes so extensively disseminated that it has been said to prevail after the manner of an epidemic.

Although this parasite rarely causes death, still it is often the cause of great distress and loss of strength to regiments quartered in those places where it is endemic.

In the *Statistical Sanitary and Medical Reports of the Army Medical Department* for 1860, the admissions for *Dracunculus* into the hospital may be shown as follows:

#### I.—EUROPEANS.

STATIONS.	Average Strength.	Total Admissions.	Ratio per 1000.
Home Stations,* . . . . .	97,708	28	3
Mauritius, . . . . .	1,886	17	9.0
Bengal, . . . . .	42,371	51	1.2
Madras, . . . . .	10,696	19	1.7
Bombay, . . . . .	11,388	114	10.0

\* It is of course to be inferred that these men had served abroad in countries where *Dracunculus* is endemic.

## II.—BLACK TROOPS AND ASIATICS.

STATIONS.	Average Strength.	Total Admissions.	Ratio per 1000.
Sierra Leone, . . . . .	879	1	2.6
Gold Coast, . . . . .	313	77	246.0
South China, . . . . .	2611	73	26.0

It would be interesting to know how long each man was off duty or under treatment for the disease induced by this parasite. In India the average number of days which those affected with the worm remain in hospital increases progressively with advancing years. During the *first period* (18 and under 20 years of age) the average number of days under treatment—during which period each person was rendered ineffective—was 14.8; during the *second period* (20 and under 25 years) it was 16.188 days; during the *third period* (25 and under 30 years) it was 18.001 days; during the *fourth period* (30 and under 35 years) it was 22.718 days; during the *fifth period* (35 and under 40 years) it was 24.290 days; during the *sixth period* (40 and under 45 years) it was 31.620 days (EWART).

Dr. Leith, in the *Bombay Mortuary Reports*, records 133 deaths from *Dracunculus* in eight years (from 1848 to 1857). A fatal result generally takes place from hectic (LORIMER) and exhaustion, consequent on the copious discharges which sometimes follow the presence of the parasite, or from abscesses forming and bursting into the abdominal cavity (EWART). Death has followed from tetanus (Drs. Minas and McKenzie, *Trans. of Hyderabad Med. and Phil. Society*). Great destruction of tissues sometimes results from sloughing; and deep-seated inflammation may attend its existence, with the formation of abscesses and deep-seated sinuses. The death of one person is recorded by Dr. Minas at Sirsa, in whom the whole body and skin was a network of Guinea worms. As a rule, however, the patient is unconscious of the presence of the *Dracunculus* till it is matured and ready to make its exit.

**The Number of Worms** observed in any one individual is very various. In the majority of cases only one is present, or known to be making its exit at one time. But there are remarkable exceptions to this rule. Mr. Forbes mentions that most of those affected have had two worms extracted; but many have had four, five, and six; and when he wrote he was then treating a man in hospital in whom no less than fifteen were exposed to view, and many of these were extracted. Dr. A. Farre mentions that as many as *fifty* worms have been met with in one person. Such cases, however, are confessedly rare even in India, where *fifteen* worms is about the greatest number observed.

**Seat or Locality of the Parasite.**—The lower extremities are by far the most frequently affected—or rather, the parasite most frequently tends to make its exit there;—98.95 per cent. of the parasites do so.

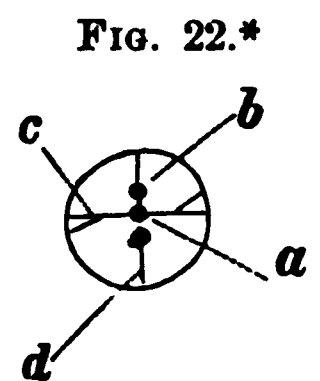


Medical Service, relates a very distressing instance of this kind which happened to an officer who had already extracted a Guinea worm fifteen inches long from his scrotum. Ten days afterwards he experienced an unpleasant sensation in the posterior aspect of the left thigh. Day by day the sensation shifted lower down, till it reached the popliteal space. A few days later the sensation was experienced in the calf. Hitherto nothing was visible; but at the end of sixteen days from the first sensation in the thigh the convolutions of a Guinea worm could be distinctly traced at the outer side of the ankle-joint. Dr. Stewart now wished to cut down and extract the parasite, but the evening was too dark, and he delayed till the following morning. By the morning visit, however, the parasite had again fled, and had taken up a position in the deeper muscles of the foot. Not a trace of the worm could be recognized in the place which he had evacuated. Many abscesses now formed, and severe inflammation of the foot resulted, which confined the patient for three months before he was free of this wandering parasite. Dr. Ewart says he has seen the worm change its position from the upper part of the lateral aspect of the thorax to the groin in the course of twenty-four hours; but he has never seen the creature travel from below upwards (*Indian Annals*, vol. vi, p. 490, July, 1859).

**Structure of the Dracunculus.**—It is often a matter of extreme difficulty to extract the worm without breaking it, and, on account of its remarkable elasticity (for it may be extended to twice its apparently natural length), good measurements of any large number of worms are not easily obtained (Busk).

Of forty Indian specimens Ewart gives the average length at 25.25 inches, the shortest being  $12\frac{3}{4}$  inches, the longest 40 inches. Clot Bey records their length at from 6 inches to 4 feet, in Egypt. Carter gives their dimensions in India at about 28 inches long, and  $\frac{1}{4}$ th of an inch in diameter. He has dissected five. Busk gives the dimensions at from 4 to 6 feet, and  $\frac{1}{12}$ th of an inch in diameter; and he has made out that it grows in the human areolar tissue at the rate of about an inch a week. H. C. Bastian, Esq., of University College, has recently read an account of the anatomy of this parasite at the Linnæan Society; and from the records of these excellent observers we have now a very complete account of the anatomy of the Guinea worm.

The anterior end of the worm (Fig. 22) may be recognized by a "punctum" in its centre,  $\frac{1}{200}$ th of an inch in diameter, surrounded by rugæ in circles, the external of which was  $\frac{1}{10}$  of an inch in diameter. Above and below are two papillæ opposite each other, with a transparent area in the centre of each. These are rather



\* Diagram of the head or anterior end of the Guinea worm; showing (a.) Punctiform mouth  $\frac{1}{200}$ th of an inch in diameter; (b.) Upper large papillæ; (c.) One of small lateral papillæ; (d) One of four crucial white lines meeting at the mouth, and occupying intermuscular spaces (H. C. BASTIAN, Esq.)

oval,  $\frac{1}{4}$ th of an inch in diameter, with a transparent area of  $\frac{1}{10}$ th of an inch. Besides these, two lateral tubercles exist, much smaller, more indistinct, and further from the punctum than the upper and lower papillæ. They are  $\frac{1}{10}$ th of an inch in diameter.

It is difficult to obtain a good view of the head; for, as it is the first part to protrude through the skin, it is usually rubbed off or destroyed by the treatment adopted for extraction.

Great varieties in form are presented by the tail or posterior end of the worm (Fig. 23). The remains of the attenuated extremity of the young *Filaria*, being more or less persistent in the form of a hook or spikelet, was believed at one time to be the penis of a male; and such specimens as showed such spikelets have been mistaken for male Guinea worms. All these forms, as Busk showed, have been found in specimens containing living young ones (proligerous). All are females that have yet been found, and no males are known to exist in the human body. The strength of the tissue of the *Dracunculus* is such that a loop of the parasite

FIG. 23.\*



will suspend a weight of 11½ ounces (SCOTT), and it is elastic to a remarkable degree. On opening the body, two longitudinal muscular bands are seen on the dorsal and two on the ventral aspect, running from end to end; while circular or transverse rugæ mark the whole extent of the worm; and these are approximated or apart as the worm is contracted or extended. The body of the worm (Fig. 24) contains an alimentary canal, which commences at the "punctum," and terminates in the concavity of the tail end. It is of a yellow color, nearly uniform in size throughout its extent, and in its course through the body winds several times round the genital tube (BASTIAN). No outlet has yet been detected. It is distinct from the tube containing the young (FORBES).

The genital organs consist of a large uterine sac or tube, occupying nearly the whole length of the worm, and terminating abruptly at either extremity in a much smaller tube (probably ovarian), about three-quarters of an inch in length. No vagina or vulva can be discovered (BASTIAN).

The whole extent of this uterine sac or capsule is crowded with

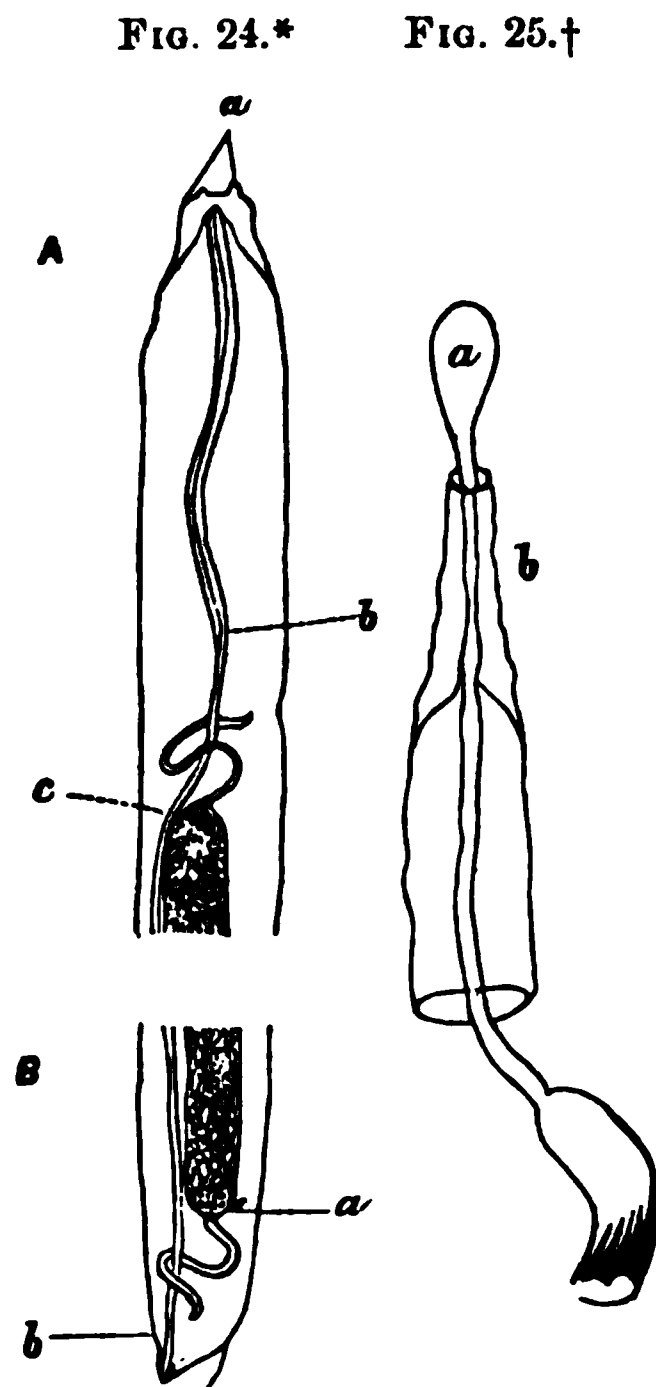
\* Various forms of the caudal end of the Guinea worm.—(A, B, C) after BUSK—all of them proligerous; (D) after CARTER; (E) after GREENHOW.

innumerable young, and, with the exception of a transparent half inch or so of the worm, the whole extent of the parent seems to be a *uterus*, a *matrix*, or a *proligerous capsule*, carrying a countless offspring, to which no parturient female of any animal can be compared for productiveness; and from the fact that no inlet has ever been discovered to the genital organs, and from various circumstances, Mr. Bastian has endeavored to show that this innumerable progeny has been produced by a process of *parthenogenesis* similar to that with which we are so familiar in the *Aphis*.

If a living worm recently extracted be well lit up by an argand lamp, the hair-like filaments may be seen in motion with a good simple lens; and if a section be made across the parasite after it has been hardened in glue, the young may be demonstrated *in situ* (Fig. 26).

When the animal is mature, and presenting its head through the skin, it protrudes the extremity of the proligerous capsule through one of the small papillæ or puncta, carrying forward a prolongation of something in the form of a loose corrugated sheath (Fig. 25). It gradually assumes the form of a dilated vesicle filled with limpid fluid—the contents of the proligerous capsule—containing flocculent granular matter and young Guinea worms. Carter tells us that, if kept moist, the full-grown parent will live many hours; and in this state the young will live till the parent begins to decompose; and when the head end of the worm during its extraction may have been dried up for several days outside the wound, the remaining part with the young still remains alive. Mr. Busk says that the young survive after having undergone a considerable degree of drying up.

**Description of the Young.**—They are exceedingly numerous, and constitute the bulk of the contents of the parent's body; but are less numerous towards the tail end. Each young one may be said to consist of a body and a tail, hair-like and finely pointed. The

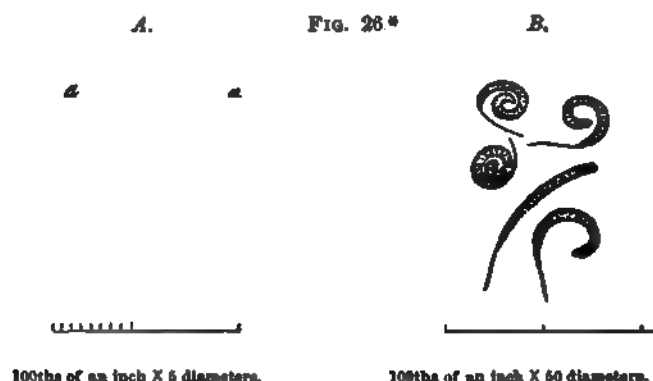


\* A.—Anterior extremity of worm, slit open and magnified, showing, (a.) Upper and lower cephalic papillæ in profile; (b.) Junction of œsophagus with intestine, and constriction of peritoneal sheath; (c.) Anterior termination of uterus, with short ovarian tube. B.—Posterior extremity of worm, slit open and magnified in same way, showing its hook-like termination; and, (a.) Posterior termination of uterus with ovarian tube; (b.) Termination of intestine (BASTIAN).

† Anterior extremity. The ovisac (a.) is protruded, dilated, and contains young; (b.) A funnel-shaped sheath surrounding the protruding ovisac (GREENHOW).



body constitutes  $\frac{3}{4}$ ths, and the tail  $\frac{1}{4}$ th of the whole length. The anterior extremity has a blunt end, with a rounded oval orifice



communicating with a cavity occupying about one-half of the whole length of the body, and terminating cœcally.

**Symptoms and Life of the Guinea Worm.**—As a parasite in the human body it may be studied during two periods of existence; but from the beginning to the end of its cycle of development its history embraces at least *three*, if not *four*, phases of existence or forms of life: (1.) During the first period of its existence in the human body the Guinea worm parasite is latent, residing in the connective tissue, at variable depths from the surface. During this period it does not exert any irritating influence on the surrounding tissue, as has been shown by dissections (BUSK). (2.) The second period of existence comprehends that of ripening or maturation of the worm and its progeny, when the worm makes itself felt, and begins its exit through the skin. This period is marked by characteristic symptoms. Drs. Scott, Forbes, Morehead, Lorimer, and Van Someran all agree in stating that the earlier symptoms are a pricking itching heat, which is felt at the part where the worm exists, seldom amounting to pain till after the lapse of three or four weeks. A small vesicle forms over the part, which immediately precedes the appearance of the anterior end of the worm. Dr. Scott was himself a sufferer, and writes feelingly on this point. (See *Med.-Chir. Review*, 1823.) This itching may happen before any vesicle forms; and when the vesicle forms it rapidly enlarges—so rapidly that in a few hours it attains the size of a good large filbert (LORIMER). If this vesicle is opened early, it is seen to contain a clear and limpid fluid (the fibrinous serum of irritation?); but if untouched for a day or two, its contents become turbid, and sometimes bloody, from the rupture of the proligerous sac, and the dis-

\* A.—Appearance of transverse section of adult Guinea worm, as seen throughout the greater part of its length.—(a, a, a, a.) Sections of the *four* longitudinal muscles; (b.) The intestine flattened, and lying along the edge of one of the longitudinal muscles; (c.) Walls of the uterine sac, often adherent to the parietes of the body. B.—Young of the Guinea worm more or less spirally curved (BASTIAN).

charge of the young *Filaria* amongst the serum. These greatly add to the irritation, so much so, that when the cuticle is removed, an angry-looking ulcer is exposed, in the centre of which the parasite may be seen presenting itself, *with a thin transparent tendril about an inch in length hanging from its point.*

After the appearance of the vesicle or blister, it is sometimes weeks before the worm protrudes itself.

The contents of the blister, when turbid, are a discharge from the tube of the animal; as Wilkins, of 4th Light Dragoons, first surmised, and as shown afterwards by the independent observations of Forbes, who found that the best way to procure the young Guinea worm for microscopic examination was to lay open this vesicle before the delicate membrane of the proligerous capsule burst. After the escape of the serum from the vesicle, the delicate transparent membranous tube or *cul de sac* is sometimes protruded from the extremity of the worm; and if cold water is *gently* poured in a *constant stream* upon this protrusion, the dilatation and protrusion increase, till an innumerable quantity of young is ejected from the ruptured orifice of the dilated tube. Forbes says that he has often repeated this experiment; and in one instance the transparent tube was again retracted within the limb, after three emissions of young Guinea worms. On the following day the tube was found again protruding as before; and the same result (namely emission of young) followed the gentle application of the stream of water. The animal will emit its young daily in this way for some time; and when it ceases to emit them, it is then time to begin the extraction of the parasite (FORBES).

**The Period of the Year when Dracunculus is most Prevalent.**—This seems to vary considerably in different parts of India, and the probable causes of these differences are of great interest in regard to the origin and spread of this parasitic affection.

At Madras and its vicinity Guinea worm annually appears with greater prevalence during the hot season (LORIMER), comprehending February, March, April, May, and June. At Dharwar and its vicinity the admissions to hospital for Guinea worm generally commence in April and May. At this time water is scarce, every tank is dried up, wells yield a scanty supply, and the natives are obliged to remain at the bottom of the wells by turns, till the required supply is obtained; and when the monsoon sets in (rainy season), the admissions gradually increase through June, July, August, and September. The increase of the disease amongst soldiers or residents seems to advance with length of residence, generally during the rainy season. In the Bombay and Matoongha districts the admissions to hospital begin in May or June (irrigation of fields by the natives being common at this time), but it chiefly prevails during the rainy months of June, July, August, and September, and is rare after October (SMYTTAN). Dubois, a missionary at Sattimungalum, says that its annual endemic prevalence in the Carnatic villages is in December, January, and February, during which time more than half the inhabitants are affected. Dr. Morehead's experience at Kirkee and vicinity gave March, April, May, June, and July, as the months of gradual increase and prevalence; and September,



Every regiment which has occupied the lines of the 24th Regiment at Secunderabad, "near the large tank called the 'Hausen Saughur,'" has suffered from the *Dracunculus* (LORIMER). The cause of the disease exists *in or near* the lines at that place; and the soil is marshy which borders on the tank. The experience of the 19th, the 4th, 5th, 1st, and 35th Regiments of Native Infantry, all fix the locality of the Guinea worm germs to be "*in or near these lines.*" For example, the 19th Regiment arrived at Vepery on the 20th May, 1838. It had been free from *Dracunculus* for five years before: twelve months after its arrival twenty-eight cases of Guinea worm appeared, and several cases amongst the followers and children. The 45th Regiment occupied the same lines previous to the arrival of the 19th Regiment; and the disease appeared amongst them at the same season of the year, and after twelve months' residence. The Guinea worm had not been amongst them for many years before. At Perampore (in the 1st Regiment, N. I.) it manifested itself, after twelve months' residence, in March, April, and May. For many years previously Guinea worm had been unknown in the Regiment. Those who suffer most in cantonments are those who use water of the filthiest kinds.

On the authority of Scott, Smyttan, Chisholm, and Duncan, Guinea worms are said to have been found in the earth or soil, and that they have been dug out of moist earth. There can be little doubt, however, but that the worms so found were specimens of the *Gordiaceæ*.

In some form or another, therefore, the Guinea worm has an existence in moist earth and mud; and it is probable that the hair-like worms found by gardeners in India coiled up together may be the young *filaria* of the Guinea worm in sexual congress; whose progeny, as *Zoösperms*, or as filiform female worms (like the Tank worm of Carter), make their way into the body. It is known that the *Gordius aquaticus*, when young, enters the bodies of large water beetles, and at a certain stage of life it leaves its abode in the beetle and goes into the water, where it becomes a variety of *Tank* worm. It appears that there are white and brown *Tank* worms—nay, that there are no fewer than seventeen species of minute *Filaria* (CARTER, MITCHELL); and some say that all *Tank* worms are white at first, but become black after a time in the water (GUNTHER). Observations are greatly wanted on these points. According to observations collected by Pallas and quoted by Vogel, it appears that even in Europe thread worms like the *G. aquaticus*, common in stagnant water and moist earth, can in certain cases infest the human subject (*De Infestis Viventibus intra Viventia*, p. 11).

The most obscure and incomprehensible parts of the history of this parasite are—(1.) The phase of its existence and that of its young after it leaves the body of man; and (2.) The future life of the young, and their sexual differentiation.

The parasite may be removed in several ways by surgical interference—either by cutting down upon it; or, after it begins to show itself, to commence winding it on a stick, gently pulling a portion of it out every day. But there is a natural termination to all dis-



in the water with the worm hanging loose, drawing the limb quickly backwards and forwards through the water, and from side to side, till expulsion is effected." The natives do not believe that they get the parasite from bathing in the water.

In these and similar cases the parent, being carried away in the stream, finds a place to die, and so gives freedom to her immense brood of young. The water seems congenial to the parent Guinea worm, and sooner than anything else induces her to leave her position in the human body, and so to extricate herself, perhaps by stimulation of the muscular structures. This water method of extraction was also recommended by Dr. Helenus Scott, of Madras (*Edin. Med. and Surg. Journal*, vol. xviii).

**Vitality of the Parasite in Water.**—It has been stated that young *Dracunculi* die in four, five, or six days if placed in pure water from well or tank (and that is the case with many animals), simply for want of food. Water *not pure* is, no doubt, the proper element for them (MITCHELL). Those artificially kept in impalpable red clay, partially covered with water, and exposed to the sun, were found alive after fifteen, eighteen, and twenty-one days, burrowing into the fine, soft and ochry mud.

Forbes experimented on two pups five or six months old. He poured down their throats water containing the young Guinea worm *Filaria*. After three minutes the first pup became uneasy, sick, and vomited; the watery part of which was found to contain the animal still alive. Four hours after this the pup was killed, when abundance of *Filaria* were seen in the mucus of the stomach and duodenum; but none showed signs of life. The other pup was killed twenty-four hours afterwards, but none were alive, although abundant in the mucus. Lorimer tried upon himself and others if the parasite could be propagated by inoculation of the young *Filaria* emitted from the parent's orifice. Five besides himself were inoculated. He naively remarks that *he is sorry to say* they did not hatch in any, although in his own case he put them in their favorite place—namely, the *foot and ankle*.

Such experiments were not likely to succeed, from the delicate nature of the young *Filaria*, and because they were introduced under unnatural circumstances. Inflammation and pus are inimical to the life of the worm. Besides, it is most probable that they enter the body in some other form. They seem to go through another stage of existence, and become sexual; for it is *only females*, and these *impregnated ones*, which are found in the body of man. The progeny of the sexual *Filaria*—and the impregnated females only of that progeny—would therefore seem to be the *Dracunculus* of man.

Dr. Ewart, in his able paper on the vital statistics of the Meywar Bheel corps, writes as follows: "I am inclined to believe that Guinea worm is propagated by a female and impregnated *ZoöspERM*, and not directly from either the young of the full-grown female Guinea worm or from tank worms" (*Indian Annals*, vol. vi, July, 1859).

**Examination of Water, Mud, and Tanks.**—In the months of August and September, 1837, Dr. Forbes examined several of the tanks in



worm; but that persons who bathe in water in which the former is found may expect to have the latter.

“Dr. Carter further states that the Industrial School is situated near an old artillery barrack, now in ruins and overgrown with weeds, which had to be abandoned in consequence of the havoc made among all ranks, officers as well as men, by this fearful parasite” (MITCHELL, *l. c.*).

The habit of the tank worm is to bury itself under any organic *debris* that may be in the water in which it is found; and if it be disturbed, it will immediately seek a hiding-place, nor rest until again covered. This implies that its proper habitat is the bottom of tanks, wells, or other reservoirs, among the decayed and decaying organic matter. It may be assumed that the water-carriers referred to by Dr. Morehead were Army Bheesties, who as such probably had access to good puckah wells (Dr. Morehead having found that Guinea worm was not more common among them than among other people), and as the tank worm, habitually resident in the mud at the bottom, would only be disturbed when the water became very low, and would get back again to its retreat if possible, the fact of water-carriers being as little affected with Guinea worm in the upper part of the body as other people does not carry so much weight as at first it would seem to do, and as it would in reality if the tank worm was in the habit of swimming at the surface like many other aquatic animals. It has not been said that the worm finds its way into the body by any of the natural cavities of the body, such as the alimentary canal. On the contrary, it is supposed that the water may be drunk with impunity, as known by experience, and from the experiments of Forbes already noticed.

The young *Filaria* can work its way into a proper receptacle by its pointed extremity, “which is a long cone, ending in a point so inconceivably fine that the point of a cambric needle is a large marline-spike in comparison with it.” But notwithstanding its exceeding tenuity, it appears tolerably rigid, and as the proper receptacle referred to is one of the sudoriparous ducts, a ready-made aperture exists for a distance quite long enough to contain so small a creature; and it is by no means inconceivable to one who has seen its active exertions, that it should be able thus to hide itself in a foot or leg kept for some time in the water. It is unnecessary, perhaps, to do more than allude to the well-known native custom of going into a tank to take water. In these tanks water-carriers may often be seen standing for five or ten minutes at a stretch, chatting and washing themselves. They of course stir up the bottom mud, and, if the tank worm be there, and is the origin of the Guinea worm, they certainly afford it every opportunity to effect a lodgment—the instinct of the parasite directing the effort. One circumstance which makes this the probable mode of entry is that natives are much more subject to attack than Europeans.

Thus the evidence is very strong which refers the entrance of the parasite to bathing or lying on moist places where the tank worms abound.





the germs of the Guinea worm, which find their way into the sea-men of the ship, who are in the habit of going into these canoes with bare feet.

Negative evidence, which would attempt to show that tank worm does not exist, cannot be received. Most of the examinations on which such negative evidence rests have been imperfect; having been made with instruments confessedly imperfect, and perhaps by men not accustomed to use the instrument. I speak only of written and published statements, and on the authority of Dr. Lorimer.

**Problems for Solution.**—Forty years ago Dr. Scott suggested that a patient and careful investigation of soils and waters ought to be made wherever *Dracunculus* is known to be endemic, and especially the soil round brackish wells and the beds of tanks. Morehead, in 1833, recommended that the following points be attended to, namely: (1.) Geological structure of the ground and nature of the site generally; (2.) Nature of soil, wells, and well-water; (3.) Nature of rocks through which wells are sunk; (4.) Abundance or scarcity of water; (5.) Seasons of increase or decrease of the disease; (6.) Opinions of natives.

The occurrence of Guinea worm is sometimes defined by a distance of a few miles. So it is with many algæ and minute water animals and plants as to habitat.\*

2. *Filaria lentis*.—Length,  $\frac{3}{16}$ ths to  $\frac{6}{16}$ ths; width,  $\frac{1}{16}$ th of an inch. The body is thick posteriorly, filiform, and ending in a pointed tail, transparent, and partly coiled up in a spiral form. The *alimentary canal* is surrounded by the folds of the oviduct.

This *Filaria* (*F. lentis*) is very imperfectly known, and the female only has been seen. It was detected by Nordmann in the *liquor Morgagni* of the capsule of a crystalline lens of a man whose lens had been extracted for cataract by the Baron Von Gräfe. In this instance the capsule of the lens had been extracted entire; and upon a careful examination half an hour after extraction, there were observed in the fluid two minute and delicate *Filariæ* coiled up in the form of a ring. One of them presented a rupture in the middle of its body (probably made by the extracting needle), from which rupture the intestinal canal was protruding. The other was entire, and

---

\* My friend, H. C. Bastian, Esq., M.B., of the London University, London, has recently furnished a most interesting account of the anatomy of the Guinea worm to the Linnæan Society, and has been kind enough to furnish me with drawings of his observations; and he writes to me as follows: "Since I saw you last I have discovered several species of Carter's 'tank worms' in soft mud, &c. (at Falmouth); that is, small *Nematoids*, agreeing in almost every respect with those found by him in Bombay. The more I see of these, the more thoroughly am I convinced of the undoubted relationship existing between them and the Guinea worm, coinciding as they do in their anatomy even to minute details, and in many respects where there is a salient distinction between the anatomy of the *Dracunculus* and that of the *Ascarides*. One which I sketched to-day had an exsertile, rigid, sharp-pointed œsophagus.

"The great difficulty in the theory is to account for the fact of the localization of the disease, whilst these animals are probably so widely spread; and I suppose it is one particular species which is limited in its diffusion; but I suspect that many of those others will hereafter be discovered as parasites in animals or vegetables. The *Vibrio tritico* I have examined, and find it to be a worm essentially similar; and Dr. Cobbold tells me that he has found a long thread-like worm in the subcutaneous tissue of the back of a water-bird. The whole question wants working out."

measured about  $\frac{1}{80}$ ths of an inch in length. It presented a simple mouth, without any apparent papillæ, such as are seen to characterize the large *Filaria* which infests the eye of the horse; and through the transparent integument could be seen a straight intestinal canal, surrounded by convolutions of the oviducts, and terminating at an incurved anal extremity (OWEN, p. 64).

A *Filaria oculi vel lachrymalis* has been described as not uncommon among the negroes on the Angola coast, where it is called *loa*; also at Guadaloupe, Cayenne, and Martinique. Its length is  $\frac{1}{10}$ ths to  $\frac{1}{8}$ ths of a line. It is a filiform, slender worm, pointed at one end, obtuse at the other, tolerably firm, and of a white-yellow color.

The parasite has been considered a *Strongylus* by some, by others a young Guinea worm, and by others as an *Oxyuris vermicularis*.

**Treatment of those Infested by the Round Worms.**—The habitat of the *Ascarides* being for the most part a collection of mucus, the means used for their expulsion is generally some purgative medicine, as two grains of *calomel* and ten grains of *jalap*, or as many of *scammony*, given two or three times a week. The purgative ought in no instance to be given oftener; for if the purging be continued, the intestine is weakened, and more mucus secreted; so that the secretion which harbors them is increased. In weakly children small doses of Epsom salts will ultimately effect the same object, and with less distress to the patient. Many persons place great confidence in *calomel* as a medicine capable of destroying them; but it does not appear to act beneficially except as a purgative, and consequently it is an auxiliary, and not by any means the most valuable part of the treatment.

The *Oxyurides* or small vermicular *Ascaris*, being situated so near the rectum, enemata have at all times been much used in the treatment of these cases; and injections of oil have been much commended, especially of castor oil, olive oil, or sweet oil. But these animals will live from thirty-six to forty-eight hours in castor oil. Indeed, very little benefit has been derived from any such local treatment. Warm water injections tranquillize the intestine, and give more temporary relief than anything else. The *Oxyurides* are killed by cold; but it may not always be safe to throw a cold injection into the colon of a child. But if the child is otherwise a vigorous child, small injections of very cold water may be cautiously administered, with a few drops of *ether* or of *alcohol*; and injections of the following bitter substances have been found very useful in the treatment of the *Ascaris vermicularis*: Three or four ounces of a strong infusion of *quassia* repeated three or four times, or a similar quantity of *lime-water*, have been found of service. At the same time it is also well to administer internally some bitter medicine;—for example, half an ounce (or any dose suitable to the age and strength of the child) of compound decoction of *aloes*, taken in the morning fasting, once or twice a week; and three ounces (or other suitable dose) of infusion of *quassia* may be taken every morning that the *aloes* is not taken.

*Chloride of sodium*, to the extent of an ounce in a pint of *quassia* infusion, has also been found a useful injection; so also has an enema

composed of *aloes*, *carbonate of potash*, and *mucilage of starch*. But whatever local remedies are used, it is necessary to attend to the general health, which usually is at fault. The digestion is generally slow and imperfect, the secretions from the mucous membrane of the alimentary canal being abnormal. For this condition, small doses of the extract of *nux vomica*, with *sulphate of iron*, in extract of *gentian* or *aloes*, or in rhubarb or colocynth pill mass, taken twice a day, will be found of great service. Santonine may be of service in some cases.

From what has been already written, it will be seen how important it is, in the treatment of all these diseases, to take every means of utterly destroying, by burning or by chemical agents, all debris or excreta which may be passed by patients suffering from these parasites, and also how necessary it is to look well to the purity of all water supply used either for the purposes of food, drinking, or bathing, and to the quality of pork or bacon, in connection with the *trichina spiralis*.

#### ACCIDENTAL PARASITES.

Of this provisionally named class, several forms of which have been named, the occurrence of "PENTASTOMA CONSTRICTUM" in the human body as a cause of painful disease and death may be given as an example.

The author had two portions of lung and three portions of liver, each containing an unusual parasite, sent to him from Jamaica, in August, 1865, for the Museum of the Army Medical Department at Netley. Staff Assistant-Surgeon Edward Barrett Kearney, Esq., is the donor of the specimens; and from his history of the patient's fatal illness, the following account has been drawn up:

On the 11th of January, 1865, private Isaac Newton was admitted into the hospital of the 5th West India Regiment, at Up Park Camp, Jamaica, for an attack of *tonsillitis*. He was an African, enlisted about eight months previously from the slave depot at Rupert's Valley, St. Helena, where all slaves captured in slave-ships are kept until disposed of. He appeared to be about twenty-one years of age, and of a thin, spare habit of body.

On admission the tonsils were inflamed and enlarged, but not ulcerated; and there were aphthous ulcers about the tongue. He suffered from headache and pain across the back.

On the morning of the 14th he complained of great pain in the abdomen, which became tympanitic. His tongue was clean, but vividly red at the edges and tip, and it felt dry to the touch. The skin was very hot and dry and harsh, and his pulse 100. The pulse continued to increase in quickness; sordes soon began to appear about the mouth and teeth, and the tongue became furred and cracked. Large moist crepitation was heard over the whole surface of both lungs. He became low, and disinclined to be spoken to, and by six o'clock in the evening his mind appeared to be confused. He passed his urine and his stools involuntarily in bed.

On the 15th there was no improvement in his condition, and at ten o'clock at night he appeared to be in much the same state as before, and the bowels were confined.

On the 16th he appeared livelier in the morning, the skin cooler, but still dry. He was thirsty, and sordes were still about the lips and teeth. The conjunctivæ of both eyes were stained of a vivid yellow color.

About nine in the evening he became suddenly worse. His pulse became very weak and almost indistinct, the skin cold, the countenance sunken, and covered with a copious perspiration. He appeared to be sinking, and he died at half-past ten that night.

*Post-mortem Examination Fourteen Hours after Death.*—The general appearance of the body was that of emaciation, with yellowness of the conjunctiva.

*Thorax.*—The subcutaneous areolar tissue over the chest and abdomen was of a deep yellow color. The *pericardium* contained about four ounces of deep amber-colored fluid.

The *Heart* was large and pale, but its substance was otherwise normal, and its valves healthy. The *Lungs* were both highly congested; and when cut into, a bloody frothy fluid exuded in quantity. The substance of both was very friable and yellowish in color. "On the anterior surface of the right lung and near the edge of its lower lobe, one or two yellow specks appeared. They were about the size of a spangle, and when cut into, worms were seen regularly encrusted in its substance." On the posterior surface of both lungs there were numerous adhesions of long standing.

*Abdomen.*—The *Liver* was very large, extending into the left hypochondrium. "Its surface was dotted over, both posteriorly and anteriorly, with about twenty or thirty yellow specks similar to those seen in the lung." The hepatic substance appeared paler and rather more soft than natural.

*Stomach.*—It was distended with air and fluid, containing about a pint and a half of a dark-green colored fluid. The mucous membrane was congested in patches, in the stomach, and along the whole tract of the intestines. There was no ulceration, and no appearance of *Tæniæ*, either continuous or in proglottides, could be discovered. Other organs were healthy.

*Description of the Parasite and the Lesions it Produced.*—Fig. 27 represents a small portion of the lung, with the little worm, seen at

FIG. 27.

*a*, curled up in its cyst. The pleura has been removed, so as to expose the "rings," "markings," or "constrictions," which are characteristic of the body of this parasite. The pleura was opaque, and considerably thickened, probably from the irritation of the parasite.

The appearance of the parasite on the surface of the liver was exactly similar to that in the lung, and therefore it is unnecessary to give another drawing; and wherever the serous covering

of the organ was sufficiently transparent, the constrictions of the parasite could be seen distinctly shining through.

Fig. 28 represents two specimens of the parasite removed from their cysts. They are of the natural size, and one of them, *a*, is much shorter than the other, the constrictions being closer together: *a* measures five lines in length, *b* measures about eight lines. In diameter they are about one line. About 20 to 23 rings or constrictions can be counted on the elongated body, at tolerably regular intervals, and somewhat spirally arranged.

FIG. 28.



Fig. 29 represents the two specimens of the parasite slightly magnified (about three diameters): *a* is the shorter; *b* and *c* are the posterior and anterior aspects of the longer of the two worms. The head end appears compressed, so as to be flat and square-shaped at the end. It is seen to be marked with five spots on the anterior aspect, as shown at *c*. The posterior aspect of the flattened head, as shown at *b*, is comparatively smooth. The elongated body is rounded, and the caudal end terminates in a blunt-pointed cone. The constrictions appear like folds of the outer covering of the worm, each fold overlapping the one which follows, from the head to the tail. The body of the parasite is rounded, and not flat, as the tape-worms or cysticerci.

FIG. 29.

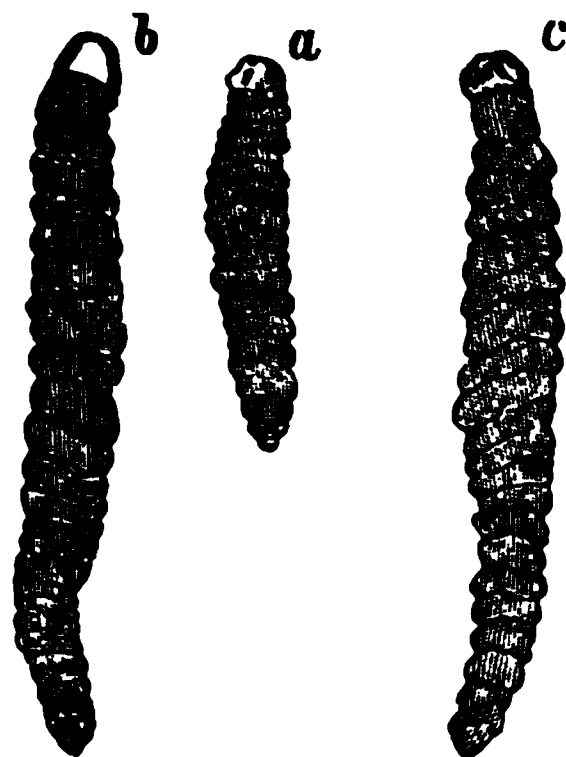


Fig. 30 represents the anterior aspect of the flattened head end (*cephalothorax*) of the parasite. It is so highly magnified as to show the nature of the five spots or marks shown in Fig. 29.

The dotted lines from *a* and *b* point to two pairs of hooks or claws—one pair on each side of a pit or mouth, *c*. The points of the claws indicated by *a* are seen nearly in profile; those at *b* are directed more towards the observer. These claws appear to be implanted in socket-like hollows or depressions, surrounded by much loose integument. These socket-like hollows appear to be elevated on the summit of the mass of tissue which lies underneath the folds of integuments surrounding the base of the hooks. These parts are regarded as the feet of the parasite, and the hooks are the foot claws.

The pit or mouth (indicated by the dotted line to *c*) is of an oval shape, the long axis of the oval lying in the direction of the length of the worm. The lip or outer margin of the pit is marked by a well-defined thin line. There are no spines nor hooks on the integument of the elongated body.

From the description and the drawings here given it will be seen that the parasite corresponds in its specific characters with the larval condition of the "*Pentastoma constrictum*." It belongs to the family *acanthotheca* of Diesing, and has no structural connection with





mined accurately the nature of the parasite he observed; and he also subsequently found two specimens of the worm preserved in the Pathological Museum at Bologna, which had been removed from the human liver (COBBOLD). Bilharz has since repeatedly detected in the livers of negroes at Cairo the parasite discovered by Pruner in 1847. Bilharz and Von Siebold made this parasite the subject of careful study; and they recognized in it a variety of *pentastoma* quite different from that which prevailed in some parts of Germany. They gave this new variety the name of *Pentastoma constrictum*—the parasite which has proved fatal in the case whose history Dr. Kearney has sent to me from Jamaica. It is the form of *pentastoma* endemic in Egypt, and hitherto it has only been found in the African negro. It differs from the *Pentastoma denticulatum* (the larval form of the *Pentastoma taenoides*), "in not being furnished with any integumentary armature of spines, and in its being a much larger worm" (COBBOLD, p. 402). The *Pentastoma constrictum* seems to be from eight to twelve times larger than the *Pentastoma denticulatum*, and therefore is all the more dangerous from its actual size (nearly an inch long); and when it occurs in great numbers, as in the present instance, it cannot fail to prove an extremely irritant "foreign body," when it escapes into a serous cavity like the pleura or peritoneum—a mode in which it seems to cause death. The latter parasite (*P. denticulatum*) has been fully described by Frerichs, and figured by him in his *Atlas*, plate xi, fig. 9, as endemic in Germany in the human liver—in which organ it is considered to be far more common than the echinococcus.\* Frerichs, however, regards the *pentastoma* endemic in Germany as devoid of clinical importance, because it does not give rise to any functional derangement. Not so, however, is such the innocent history of the *Pentastoma constrictum* as it affects the Negro; and after the history of the case now given, the clinical importance of this parasite cannot be disregarded.

As to the mode in which it tends to cause death, the evidence in this case, from symptoms and post-mortem examination, seems to point to *pneumonia* and sudden collapse from *peritonitis*. The author is able also to verify this point in the pathology of this parasitic disease still more clearly from a preparation which has been in the Museum of the Army Medical Department since 1854, but the nature of which he could not understand till the history of the case now published was so thoughtfully furnished by Dr.

---

\* "In Germany," says Frerichs, "the *pentastoma* was first found in the human liver by Zenker, in 1854: it occurs, however, not only in this gland, but also in the kidneys, and in the submucous tissue of the small intestine (WAGNER). The parasite is by no means rare with us. Zenker, at Dresden, succeeded in finding it 9 times in 168 autopsies (or, according to Kuchenmeister, 80 times in 200 autopsies). Heschl, in Vienna, met with it 5 times out of 20 autopsies; Wagner, at Leipzig, once in 10. According to Virchow, it is more common in Berlin than in Central Germany. During six months at Breslau, I (Frerichs) met with it in 5 out of 47 dead bodies. As a rule, there is only one present; in rare cases there are only two or three. It presents the form of a somewhat prominent nodule, from 1 to 1½ lines in length, which is formed by a firm, fibrous capsule, easily detached from the surrounding parts. The animal lies coiled up in the interior of this capsule" (*On Diseases of the Liver*, vol. ii, p. 276).





Assistant-Surgeon Dr. Humphrey C. Gillespie, from the preparations which are now in the Pathological Museum of the Army Medical Department at Netley.

LARVA OR GRUB THE EXCITING CAUSE OF BULAMA BOIL.

My friend Dr. Albert A. Gore, Staff Assistant-Surgeon on the West Coast of Africa, has kindly favored me with the following account of this parasite and the disease which it induces:

"This small larva or grub is of a white color, a line or two in length, and is the exciting cause of a boil occasionally seen in the Island of Bulama and its neighborhood (Fig. 32). When magnified under a low power (Fig. 33), it appears to be divided into a series of joints, and covered with minute bulbous hairs. On the anterior division are placed four or five red spots (*b*), and from either side project two hollow suction tubes (*a, a*). The posterior extremity seems to be terminated by a blunt hook. In applying a higher power, the bulbous hairs turn out to be a number of beautiful black hooklets (Fig. 34), which have a very pretty appearance on the white surface. A faint outline of a central cavity can be discerned. The hooklets are directed anteriorly.

"**Symptoms, Treatment, &c.**—Attention is first attracted to the part by feeling an extreme itchy sensation. On examination, a small red pimple is seen. After awhile a small serous discharge oozes from its centre, which sometimes seems to pulsate. If allowed to progress, it becomes a regular inflamed boil, very painful, and often causing an erysipelatous blush, with inflammation in the neighboring lymphatics, and tenderness of the glands to which they run. On the evacuation of the small abscess in the boil these symptoms gradually disappear, but a persistent red mark remains at the original seat of the disease. The treatment consists in poulticing until the little animal appears, when it can be withdrawn. If allowed to suppurate, it must be treated in the usual way of a boil under similar circumstances. The natives put in a mixture of salt and palm oil, which takes out the little grub.

"**Etiology and Pathology.**—This little worm cannot be the *chigoe* or *Pulex penetrans* of the West Indies, although it may result from the ova deposited by some similar aphanipterous insect. The *chigoe* chiefly attacks the toes or intervals between them, and causes a series of painful ulcers. It is also of a black color. This small grub is white, causes a boil or two in any portion of the body, most commonly in the thigh, arm, or abdomen; it is sporadic, although endemic. They have one symptom in common—viz., the extreme itchiness. But this is complained of in nearly every case of disease resulting from insect or other living organisms."

FIG. 32.\*



FIG. 33.†

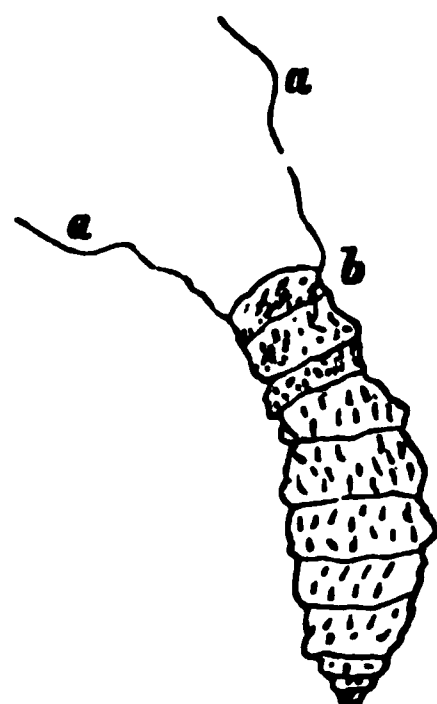


FIG. 34.‡



\* Natural size of larva or grub, the exciting cause of Bulama Boil.

† The same grub magnified by a low power: (*a, a*) Hollow suction tubes; (*b*) Fine red spots. The other dots on the body are hooklets.

‡ Two of the hooklets highly magnified. These drawings were furnished by Dr. Gore.



parasitic habits causing it to resemble some of the numerous forms of the *Crustacea*. It is not known to cause disease.

The parasitic lesions and diseases with which these *epizoa* are associated may be described as follows:

#### LOUSINESS—*Phthiriasis*.

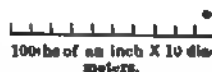
**Definition.**—A morbid state in which lice develop themselves to such an extent that a pruriginous eruption is produced (*Prurigo pedicularis*). The skins of persons liable to constitutional skin diseases in which watery or secreting eruptions (such as *eczema*) prevail, are those most favorable for the development of lice.

**Pathology.**—Five forms of lice infest the skin of man. One variety is met with on the hair of the head—the *Pediculus capitis*; a second variety infests the other hairy parts of the body, but especially the pubis; and hence its name—*Pediculus pubis*; the third form lives on the general trunk of the body—the *Pediculus corporis*; a fourth is the *Pediculus palpebrarum*; and a fifth the *Pediculus tabescentium*.

The first four species, although they live in close proximity to one another, yet strictly limit themselves to the regions mentioned. In a clinical point of view the *P. corporis* is the most important.

1. *The Body Louse* (Fig. 36) is of a whitish color, and varies from half a line to two lines in length; the body elongated and the abdominal portion broad, its margins lobulated and covered with little hairs. The thoracic portion is very narrow, and carries three legs on each side. The legs are hairy, jointed, and terminate in claws. The insect secretes itself amongst the folds of the clothing, and causes extreme itchiness of the skin where it comes to feed. Between the irritation of the insect, the debris of its exuviae, and the scratching of the skin by the patient, *papulae* arise, the summits of which being torn off, give rise to a *pruriginous eruption*, which may even become pustular. This eruption is met with most frequently on the neck, back, and shoulders, and round the waist, the parts most tightly embraced by the clothing; and where the clothes are most frequently gathered into folds, between which the lice are embedded, and where they deposit their eggs, or egg capsules, which are crystalline, shining, yellowish, opaque bodies. The lice seem to multiply fastest where eruptions such as *eczema* prevail. A case is reported by Mr. Bryant as having occurred in Guy's Hospital, in which the whole of the body was literally covered with lice. The patient had been a governess, about thirty years of age; and the irritation was so great that excoriations and scabs were produced. On admission into hospital she was put into a warm bath,

FIG. 36.\*



\* *Pediculus corporis*—female (after ANDERSON).

her clothes were destroyed, and every precaution taken to remove all the insects; but in two hours afterwards her body was again covered with lice, although she had been laid in a clean bed. She was again thoroughly washed, but the vermin reappeared immediately. Some of the insects and their ova no doubt remained adhering to the skin, hidden amongst the scabs of the excoriations; and they are known to multiply with a rapidity proportioned to the favorable nature of the soil afforded by the morbid condition of the skin of the affected person (ANDERSON, *l. c.*, p. 108). Bernard Valentin relates the history of a man, forty years of age, afflicted with insupportable itching over the whole body, and having his skin covered with little tuberosities. The physician, unable to assuage the itching, made an incision into one of the small tumors, and gave exit to an enormous quantity of lice of different forms and sizes. The same operation was performed on the other tumors with a similar result (quoted by ANDERSON, p. 110). The following instance is related by Dr. Whitehead, in his work *On the Transmission from Parent to Offspring of some Forms of Disease*, p. 173, and quoted by Dr. Tanner: A farmer, forty-three years of age, strong, and of sanguine complexion, contracted syphilis in April, 1840. Seven months afterwards he suffered from secondary symptoms. At the end of 1841 he became so annoyed by the presence of lice about

FIG. 37.\*



his person, chiefly on the trunk, that he sought again medical aid and advice. He was scrupulously clean in his habits, and had never before been troubled with these vermin. They increased in number, and produced such mental distress that fears were entertained for the integrity of his intellect. An examination of the skin showed a multitude of irritable-looking points on the front and sides of the chest, from which nits could be detached by lateral pressure. At this period the generation of the lice was so considerable and rapid that a flannel vest put on clean in the morning was crowded with them by the end of twenty-four hours. The usual remedies

had only a temporary effect, till *iodide of potassium* and *prussic acid*, taken internally, seemed to render the system unsuitable for the further development of the vermin.

2. *The Head Louse* (Fig. 37) is considerably smaller than the body louse just described. Its legs are larger in proportion to the size of the body than those of the *P. corporis*; and the abdomen is more

\* (a.) *Pediculus capitis* (male); (b.) Trachea and stigmata; (c.) Antenna (after ANDERSON).

distinctly divided into seven segments, separated from each other at the margins by deep notches.

They propagate with astonishing rapidity, and by their irritation produce an *eczema*, from which the fluid exudes abundantly, and crusts are formed, involving the cuticular debris or exuviae of the lice and the remains of epidermis. The hairs become glued together; partly by the fluid from the *eczema* and partly by the secretions of the insects as they deposit their ova in the capsules which they fix to the hairs (Fig. 38). These capsules are commonly called *nits*; and they adhere with great tenacity to the hairs.

FIG. 38.\*

FIG. 39.†



100ths of an inch X 10 diameters.

100ths of an inch X 2.0 diameters.

3. *The Crab Louse* (Fig. 39) has a shield-shape, and a much broader body in proportion to its size than either of the other two forms of lice; and there does not appear to be any distinct separation between its thorax and abdomen. It has been met with on all the hairy parts of the body except the head, but more especially on the hair of the *pubis*. It does not run about like the other lice, but grasps the stems of the hairs with its fore legs, and adheres so firmly that it is difficult to remove it without pulling out the hair. The *nits* or egg capsules are attached to the hairs in the same way

\* *Nit*, or Egg Capsule of the Louse, fixed to (b) a hair, by the glutinous secretion (c, c, c, c).

† The Crab Louse (after ANDERSON).

as on the head. Pruriginous or eczematous eruptions, which may become pustular, are the results of their existence (ANDERSON).

**Treatment** consists in the destruction of the insects and soothing the irritation. To accomplish the death of the parasites the following substances are efficient—namely, *sulphur*, *mercury*, *staphisagria*, *sabadilla*, *pyrethrum*, the *essential oils*, and *alcohol* (ANDERSON).

*Sulphur* is used in the form of vapor baths or fumigations, or the simple or compound sulphur ointment of the pharmacopœia.

*Mercury* may be employed as simple mercurial ointment, or by fumigations with *cinnabar*, or in solution of the bichloride, in the proportion of two to three grains to an ounce of water, to which some alcohol has been added.

*Staphisagria* is employed as an ointment in the proportion of an ounce of *staphisagria* with four ounces of lard, or an infusion of *staphisagria* may be made with vinegar.

*Sabadilla* may be used in powder or as an ointment; an ounce of lard being used to incorporate a drachm of *sabadilla*.

The hair should be cut short when lice infest the head; and a lotion of the *bichloride of mercury*, or some of the ointments above named, applied at once to cause the death of the insects. The *nits* may be dissolved away by *alcohol* or *dilute acetic acid* (HEBRA). The scalp should afterwards be repeatedly washed in warm water with soap, and the eruptions treated according to their nature.

Sometimes the nits and the debris of the lice are involved in the substance of these eruptions, so that care must be taken to kill any insects that may remain and be so hidden.

With regard to body lice, it is necessary either to destroy the clothes, or to expose them to a temperature of at least 150° Fahr., by steaming them, or ironing them over with a sufficiently hot iron, or to boil them. In extreme cases, such as those related at page 880, it has been found that a mixture of iodide of potassium and prussic acid in full doses cured the diseased state of the system which favored the development of lice in such numbers. After sixteen or eighteen doses, in the case recorded by Whitehead, the cure was permanently completed.

The *Pediculus pubis* is best got rid of by rubbing a lotion of bichloride of mercury amongst the roots of the hairs, taking care that it is brought in contact with every insect. The application should not only be applied to the hair of the *pubis*, but to that in the neighborhood, such as that of the *scrotum*, *perinæum*, and *anus*; and the application should be continued twice a day for a week at least. Mercurial ointment is equally efficacious; but care must be taken not to induce salivation: the hairs, rather than the skin, are the parts on which the lotion or ointment ought mainly to be applied.

## SCABIES—SYN., ITCH.

LATIN Eq., *Scabies*—Idem valet, *Psora*; FRENCH Eq., *Gale*; GERMAN Eq., *Scabies*—Syn., *Krätze*; ITALIAN Eq., *Rogna*.

**Definition.**—An eruption of distinct, slightly acuminated papules or vesicles, accompanied with constant itching, due to irritation caused by the burrowing underneath the epidermis of a female *Acarus* (*Acarus scabiei* vel *Sarcoptes hominis*), for the purpose of depositing her eggs.

**Pathology.**—The full-grown itch spider or *Acarus* is of a whitish-yellow color, and is just visible to the naked eye. The female (Fig. 40) varies in size from  $\frac{1}{10}$ th to  $\frac{1}{4}$ th of a line in length, and from

FIG. 40.\*

$\frac{1}{10}$ th to  $\frac{1}{4}$ th of a line in breadth. It is of an ovoid form, broader anteriorly than posteriorly. The anterior segment carries the head and four limbs, two on each side of the head, which are set very close to it. The head projects considerably beyond the body, is of a rounded form, and marked by a central fissure provided with mandibles. The limbs are altogether eight in number, the four posterior limbs being placed about the middle of the under surface of the body. These limbs are of a conical form, tapering towards a point. They are each composed of several jointed segments; and the four anterior limbs are each provided with a stalked sucker. The extremity of each of the hind limbs terminates in a long curved hair; and several short hairs spring beside the root of each sucker on the anterior limbs. The body is marked by numerous regularly disposed wavy lines; the dorsal surface is convex, provided with

\* *Acarus scabiei*—female (after DR. T. ANDERSON).



numerous little angular spines and little round tubercles, from each of which also springs a short conical spine. From each side of the body two hairs project; and four project posteriorly; so that, including those springing from the hind legs, the posterior half of the body is provided with twelve long hairs.

The male *Acarus scabiei* (Fig. 41) is considerably smaller than the female, and the innermost pair of posterior limbs are provided with

FIG. 41.

stalked suckers as well as the anterior limbs; while the parts corresponding to the genital organs are very distinctly marked (HEBRA, ANDERSON).

It is now impossible to say who discovered the itch insect. Avenzoar hinted at the existence of an insect in the vesicles of itch; but Moufet, in his *Theatrum Insectorum*, first mentioned it in a particular manner in 1663. Hauptmann first published a figure of it, and represented it with six feet. Redi, Lorenzo, Cestoni, and Bonomo examined numbers of them, having removed them from the papules or vesicles of the skin. They discovered also the eggs of the parasite, and even observed their extrusion.

Morgagni, Linnaeus, De Geer, Wichmann, and Waltz confirmed these observations; but, nevertheless, the existence of the parasite up till 1812 was still called in question. About this time, therefore, a considerable prize was offered by the Parisian Academy of Sciences for its demonstration; and M. Galès, an apothecary of the St. Louis Hospital, tempted by the reward, is said to have defrauded the Academy and gained the prize (ANDERSON). His investigations are reported to have been witnessed by many members of the Institute; but, nevertheless, he managed to conceal beneath the nail of his thumb the common cheese-mite, and having opened with a lancet the pustule of a patient affected with scabies, he dexterously produced the cheese-mite from beneath his nail, pretending to have removed it from the patient (ANDERSON). Many others attempted to find the *Acarus* in the pustules or vesicles; and the circumstance of such men as Galeoti, Chiarugi, Bielt, Lugol, and Mourouval having failed in finding the animal, occasioned fresh doubts regarding its existence. Their failures arose from having followed the cue given them by M. Galès, in searching for the parasite in the papules or vesicles. Moufet had long before stated that they were not to be found in the pustules, but by their sides. Casal made nearly a similar observation; and Dr. Adams remarks that they are not found in the vesicles, but in a reddish line going off from one of its sides, and in the reddish firm elevation at the termination of this line, a little distance from the vesicle. Seventeen years after Galès' demonstrations the Academy discovered, through Raspail, that they had been defrauded; and in 1834 M. Renucci, a medical student from Corsica, showed the physicians of Paris the mode of discover-

ing the *Acarus*, which is the same method as that which had been formerly mentioned by Dr. Adams. Since that time, as Rayer remarks, the existence of the *A. scabiei* has been placed beyond a doubt; and after the demonstrations of MM. Lemery, Gras, and Renucci (all of whom showed him the method of detecting it), Rayer has been able to extract it several times himself. Raspail has given an excellent description and figure of the parasite. M. Albin Gras enters into researches as to the share it has in producing the eruption, and he instituted experiments on the *Acarus* itself, which have an important bearing on the treatment of *scabies*. The habits and natural history of the parasite have been carefully investigated by Hebra; and Dr. Anderson has given an excellent account of these investigations, from which this description of the parasite and the disease is mainly taken.\*]

[The question whether the *acarus* can be transferred to man from animals seems, notwithstanding some difference of opinion amongst dermatologists, to be resolved in the affirmative. Hardy† has expressed his belief that the *acari* in animals produce in the human subject only temporary prurigo, requiring no treatment; but Hebra states that he has repeatedly seen *scabies* transmitted from animals to man in the menagerie at Schönbrunn, and he believes that the *Sarcoptes equi*, *S. canis*, *S. suis*, *S. cati*, *S. cuniculi*, of Gerlach, *S. scabiei crustosæ*, *S. vulpis*, *S. capræ*, *S. squamiferus*, and *S. minor*, of Fürstenberg, are all varieties of the same animal. He has found the *acarus* in the camel, the Egyptian sheep, and the ferret. Küchenmeister says that the *acari* of the lion and cat may be identical with the *acarus* in the human species; Eichstädt found in the crusts of a mangy horse *acari scabiei*; and Fürstenberg has seen them in the horse, the lion, the llama, the ape, and Neapolitan sheep.]

The discovery of the male *Acarus* is claimed by several observers. According to Hebra, it was first discovered by Danielssen and Boeck in Norwegian *scabies*. According to Dr. Anderson, M. Bourgogne, the maker of microscopic preparations in Paris, claims to be the discoverer. According to Devergie, the honor is due to M. Lanquetin, a pupil of St. Louis.

[The Germans give the credit of the discovery to both Krämer and Eichstädt. The former found it in 1845, but published his description and drawings in 1846.

. Lanquetin states that previous to his discovery of it in 1851, he had seen one before in the cabinet of M. Bourgogne, who got it from an employé at the St. Louis Hospital (*Notice sur la Gale*, 1859.)]

It is the female only which burrows in the epidermis of the human skin. All the male *Acari* go free on the surface of the epidermis, where sexual intercourse between male and female *Acari* is said to take place. When an impregnated female is placed on the

---

\* [*On the Parasitic Affections of the Skin.* By T. McCall Anderson, M.D., London, 1861.]

† [Hardy, *Leçons sur les Maladies de la Peau*, Professées à l'Hôpital St. Louis. 2ième partie, 2ième ed. Paris, 1868.]

surface of the skin, it seeks a suitable spot to penetrate, and raising its head at right angles to the surface, it digs, burrows, or eats its way between the scales into the deeper layers of the epidermis, where it imbeds itself, derives nourishment, and goes through the process of parturition till she dies. Having found a suitable place, an egg is laid, and each day another, the animal penetrating a little further each time, leaving its deposited eggs to occupy the space previously inhabited by itself. The direction of the canal is oblique [but horizontal through the rete mucosum], the portion first formed being of course nearest the surface. As the old epidermis is thrown off, new layers of cuticle being formed from the deeper strata, the first-laid eggs are gradually thrust upwards to the surface, where they are finally extruded, while the recently deposited ova remain in the canal close to the parent female, whose instincts lead her to make the canal in such a way that her eggs reach the surface about the time the young ones are ready to come out of the shell. The newly hatched *Acar*i (males and females) having arrived at the surface, crawl about the skin, and enter into sexual congress. The females in due time become impregnated, and, like their parent, repeat the process of burrowing and parturition just described.

[It may either proceed at once to bury itself, or it may, after awhile, leave off, and begin again at a new spot. In penetrating the superficial part of the epidermis, it seems to have some trouble; its first position is almost vertical to the skin, and it supports and braces itself by the long bristles which project from the hinder part of its body. Once through the epidermis, its steps are rapid, and it is thoroughly buried in from ten to twenty minutes. It attacks by preference those parts of the skin where the cuticle is thinnest, and a favorite site is the mouth of a hair-follicle.]

The length of time which intervenes between the laying and hatching of an egg is said to be *fourteen* days; and as the *Acarus* is found to lay one egg daily, there are rarely more than fourteen eggs in one canal at a time. •

[The number of ova deposited by a single *acarus* varies; Hebra has seen twenty-six in one cuniculus; and Gudden\* counted fifty-one in a canal, with the parent, containing ova, at the bottom. To determine the rapidity with which they are laid, and which is said to depend on the age and degree of vitality of the *acarus*, Gudden put on the skin of an uninfested person an *acarus*, which had laid already but three ova, and which had another ready to be deposited; the subject was kept all day in a moderately warm room; at the end of five days, the cuniculus was cut out and contained eleven eggs. The *acarus* takes six or seven weeks for its development from the ovum to its being impregnated. In about three months after the skin becomes the "bearer" of a fertile mother-*acarus*, a person will generally be covered with an eruption of scabies (GUDDEN).]

---

\* [*Beitrag Zur Lehre von der Scabies*, von Dr. Gudden. Würzburg, 1863.]

The canals (cuniculi) [*sillons* of the French, *milbengänge* of Hebra] which the female *Acar*i burrow have a serpentine shape, and vary from half a line to three lines long. Hebra, and Dr. Reid, of Glasgow, have seen them three or four inches in length; and Hebra mentions that they sometimes completely surround the wrist, like a bracelet. These canals have generally a whitish dotted appearance, the dots corresponding to the ova in the canals; and at the extremity of each canal is a little whitish elevation, which corresponds to the site of the parturient or defunct female *Acarus*. This whitish elevation is generally about a quarter of a line distant from the papule or vesicle; and the skin should be cleaned before endeavoring to detect it.

[The two extremities of the cuniculus are the "head" and "tail," the first being the entrance-point of the acar, and is whiter than the "tail," where the animal may be found as a sharply defined roundish point, rather deep-seated.

The appearance of the cuniculus is, however, modified (1.) by site: as just described, it is found on the hands and feet, and elbows and knees; but on the other parts of the body these appearances are modified by the development of a vesicle or pustule beneath the cuniculus. The vesicles commonly begin at the head of the passage, but frequently extend beneath it, so that it lies on their roof. The position of the parasite is always beyond the area of the vesicle, or pustule, and when a crust is formed the parent animal is never found in it, though it may contain ova, and possibly be capable of propagating the disorder. (2.) Another modification is a white dotted line on the summit of a red elongated eminence; it is found in the neck and back, in tubercular elevations (CAZENAVE), and, occasionally, on the penis, fold of arm-pit, umbilicus, nipple, and on all parts of the body exposed to long pressure from sitting or lying (HEBRA). In infants the passages may present this form on any part of the body. (3.) In old cuniculi the "head" disappears, and a red spot with a white edge continuous with the sides of the passage is left, giving to them a retort shape (HEBRA).\*

Their color varies with their position, and the degree of cleanliness of the subject. On the hands and feet of the lower classes they are usually black; in those who wash often these parts, white; on the penis, buttock, elbow, and knee, they are generally white. The dotted appearance, Gudden asserts, is due to the breathing-holes in the roofs of the passages, which he and Bourguignon are positive exist there, and which Hebra as positively denies. Hardy and Bazin† believe them to be due to the small black feces of the acar; and Hebra says, and probably truly, that they are simply caused by dirt, which has so thoroughly penetrated that it cannot be removed by washing or friction.

The most frequent sites of cuniculi are the hands and feet. In women, the line of junction of the inner side of the foot with the dorsum is a common site (HEBRA). Acari have been extracted from the forehead (LANQUETIN,‡ HILLIER), from within the lower eyelid, from a cuniculus

---

\* [Virchow's *Handbuch der Speciellen Pathologie und Therapie*. Dritte Band, 1864.]

† [*Leçons Théoriques et Cliniques sur les Affections Cutanées Parasitaires*, par le Dr. Bazin, Paris, 1858.]

‡ [*Notice sur la Gule, et sur l'Animalcule qui la produit*. Par Eugène Lanquetin, Paris, 1859.]

which was on its free edge, in an infant (AUZIAS-TURENNE); and Hebra found a beautiful cuniculus, containing an *acarus* and eight ova, within the urethra, about a line from the meatus.]

After the death of the *mother Acarus*, [the duration of whose life is from three to four months (GUDDEN,)] the epidermis which covered in the canal gives way, as the cuticle grows and desquamates; there is then left at first a depression, or open ragged furrow, bounded on each side by a ragged edge of epidermis; and as these edges become dirty, the remains of the canal present a dirty ragged line. Besides the *Acarus* and its eggs, numerous little oval or rounded blackish spots are seen in these canals, which are supposed to be the excreta of the parasites; and after the female has once entered its canal it is unable to recede, owing to the spines on its body, which project backwards. It therefore dies in the canal when parturition is finished. The eggs of the *Acarus* vary much in size, according to their age and development; and just before the larva has burst its shell, the egg is almost as large as the male *Acarus*. In the earliest stages the egg is very small, and filled with a granular-like matter (Fig. 42, *a*).

FIG. 42.\*



FIG. 43.†

It grows in the canal; and as it increases in size its contents seem to shrink and recede from the shell, and to have a distinct enveloping membrane. The bright yellow color of the embryo contrasts strongly with the clear, almost colorless, walls of the eggshell (Fig. 42, *b*, *c*, *d*). The head and legs of the embryo soon become distinctly visible, and at last the whole form of the *Acarus* (Fig. 42, *c*, *d*). Finally, the shell bursts, and the young *Acarus* escapes, leaving its shrivelled envelope (*e*) behind. The larva or young (Fig. 43) differs from the full-grown insect in the possession of two hind limbs only, in place of four. By and by, however, it casts its coat, and then appears with eight legs; and sometimes even the full-grown *Acarus*, with its eight legs, may be seen inside of its old

\* (*a*.) Egg in the first stage; (*b*.) In the second stage, their granular contents being yellow; (*c*.) Egg in the third stage, the form of the *Acarus* becoming apparent; (*d*.) The egg in the fourth stage, the *Acarus* having broken the shell; (*e*.) Eggshell after the escape of the *Acarus* (after Dr. T. ANDERSON).

† Larva or young *Acarus scabiei*—having only two hind legs (after Dr. T. ANDERSON).

six-legged skin, and thus renders the history of its development complete.

[Gudden states that the *acarus* moults three times. According to him, the *acarus* is characterized before the first moult, not only by its six legs, but also by having only two bristles at the posterior extremity of its body, and ten of the longer spines on its back. With its eight legs it acquires also four bristles and twelve spines, and after the second moult, fourteen spines. The sexual organs are not apparent before the third moult, and the male and female differ in the number of their spines, the female retaining fourteen, whilst the male loses two, and has only twelve. While it is changing its skin it inhabits small cavities in the skin, similar to those formed by the young *acari*.

The young *acari* on leaving the shell almost directly quit the cuniculi, through, Gudden maintains, the breathing-holes in the roof, and begin to move over the body very actively, and soon imbed themselves: the passages which they form, however, are very short,—less than a line in length. Gudden says, the young *acari* penetrate the skin more deeply than the mother, and cause more irritation, their bite being often followed by a small papule or vesicle, in which exudation shows itself on the second day. Before this time, the animal has usually moved off to another part of the skin, and this makes the young *acari* difficult to detect, and the diagnosis of the disease uncertain. Gudden thinks that a diagnosis may sometimes be made, in slight cases of scabies, and in which none of the larger cuniculi can be found, by cutting off the heads of some of the papules or vesicles, and examining them by the microscope, without varnish to make them transparent. The entrance of the passage formed by the young *acarus* can then be made out; when the light comes from below the dark edge of the opening looks dark. To obtain specimens of the young animal, oil of turpentine may be rubbed over the suspected portion of the skin, which killing them, they will be found in the papules or vesicles which first appear on the part to which the turpentine was applied.]

**Symptoms.**—The phenomena of the eruption of scabies are more often papular than vesicular; and the markedness of these phenomena depend partly on the length of time that the person has been affected, the number of *Acari* developed, and the degree of sensibility of the skin. It is known to infect sheep and dogs (YOUATT); and therefore hair does not preclude its existence; but it seems to prefer delicate parts of the skin—for example, the inner surfaces of the fingers, and folds of the skin between the fingers, the wrists and palms of the hands, the penis in the male and nipples in the female, as well as the hips, the feet, the umbilicus, and axillæ.

Itching, increasing at night, first attracts attention, and is a characteristic symptom. It becomes general all over the body, and the scratching aggravates the eruption. The *prurigo* of itch is generally most expressed on the forearm, lower part of the abdomen, and the upper and inner part of the thighs. Vesicular eruption is most usual on or about the fingers and nipples of females; and pustules may be met with in children whose skin is delicate, especially on the hands, feet, and hips.

[Hebra would seem to bound the part played by the *acarus* in scabies



to that of a passive carrier of the disease. He is no believer in the migratory instincts of the animal; once embedded in the skin, according to the eminent Vienna dermatologist, it is quiet, and is transferred from one part of the body to another, or even to other persons, by the finger nails of the patient, who, scratching the papules or vesicles, tears them open, and digs out the young insects and the ova. In carrying out his crotchet, he goes so far as to say that in no case is the site of the cuniculi and that of the eruption the same; for whilst the former are chiefly found on the hands, feet, penis, &c., the eruption is most abundant on the anterior parts of the body, between the mammæ, and the knees, chest, abdomen, thighs—parts of the body most accessible to scratching, to relieve the vague itching felt. Hardy says, that most generally the initial site is the penis, and that it is transferred to the fingers by scratching. The wanderings of the young animal have been satisfactorily proved by Gudden and others, and he has shown, too, that on parts of the body where scratching was impossible, papules and vesicles have made their appearance, incontestably due to the presence of young acari at points quite distant from the spot where the mother acarus nested. It is true that there is no constant relation between the intensity of the local irritation and the abundance of the eruption and the number of acari; and in the pustular scabies of children there are sometimes but few cuniculi.

For the artificial production of the disorder Gudden proposes to extract carefully an acarus from a recent cuniculus, and then to cut off the portion of skin containing the cuniculus, and examine it under the microscope; if ova are present, it is certain that the animal will deposit others. Hebra recommends that the whole cuniculus be snipped off with a pair of scissors and applied, as a surer way.

The itch insect is sensitive to the influence of temperature, heat rendering it active, and cold making it motionless. Whenever the warmth of the skin is raised, by exposure to the sun's rays or to the fire, by the body becoming warm in bed or on exercise, its nimbleness is increased, and the consequent irritation aggravated, as those suffering from scabies so well know by the terrible itching it causes, and the relief which cooling the surface brings. As a general rule too, those parts of the body which are uncovered at night remain uninfested. Gudden mentions the case of an itch patient, a woman, who being put into a strait jacket, her hands and feet were always cold, and did not become thoroughly warm even when in bed; whilst there was a great number of acari on the trunk, none could be detected upon the hands or feet; there were a few cuniculi on the inner side of the arm, which was fixed to the body. The common site of the cuniculi, as has been observed already, is on those parts of the body most exposed to pressure. In such as sit for a long time on hard benches, as cobblers, tailors, weavers, the eruption is found on the buttocks, whilst, it is said, joiners, carpenters and bricklayers never have this part of the body affected. In women the cuniculi are found in most abundance where girdles and straps have pressed upon the skin. The skin beneath tight garters, trusses, or any band, and even a crutch, is often affected (HEBRA). The penis, and, in infants, the buttocks, are a common seat. The disease is much more frequent in winter than in summer, for in an average of eighty cases, which daily present themselves for treatment at the St. Louis Hospital, Paris, in winter, there are hardly more than ten in summer, which Hardy attributes to the poorer class sleeping and huddling more together in winter than in summer, in order to keep warm by bodily heat. Hebra, however, attaches but little importance to the influence of temperature on the habits of the acarus, or

the site of the eruption, which he refers to the irritation of the skin from pressure, or chafing.\*]

There is a severe form of scabies common in Norway and some parts of continental Europe, in which the greater portion of the skin of the body becomes thickened, the natural furrows increase in depth, the pigmentary deposit is greatly augmented, and a fine white desquamation covers the surface. Here and there papules may be seen, either with dots of coagulated blood, or with whitish crusts on their summits. In these crusts portions of *Acari* and their exuviae, excrements and eggs may be found. The hairy scalp,

FIG. 44.†

covered with a crust which adheres firmly, of a bark-like consistence and yellow color, is studded with fine openings for the hairs, which are glued together. On the under surface, and in the furrows of

---

\* [For an excellent brief of the "Recent Researches on Scabies" the reader may consult the *British and Foreign Medico-Chirurgical Review*, vol. xxxvi, 1865.—EDITOR.]

† Crust from a case of the so-called *Scabies Norvegica* which occurred in Würtzburg.—(a, a, a.) Eggs of the *Acarus* in various stages of development; (b, b.) Eggshells; (c, c.) Fragments of *Acari*; (d, d.) Female *Acarus*; (e.) Larva. The little oval irregular-shaped masses are presumed to be the excrement (after ANDERSON).



this crust, multitudes of *Acari* may be seen. Such are the main features of a case recorded by Bergh, of Copenhagen, and related by Dr. Anderson in his excellent little treatise already referred to. The itching was intolerable. The crusts were principally composed of the *Acarus*, its exuviae, excrement, eggs, and egg-husks (Fig. 44). A piece of the most superficial and dense part of the crust, less than half a line square, contained 2 female *Acari*, 8 six-legged young, 21 pieces of *Acari*, 6 eggs, 53 eggshells, and about 1030 pieces of excrement. "In the deepest and softest parts of the crusts, amongst the remains of deceased generations—partly in holes and passages, partly between particles of the crusts, partly on their free surfaces, masses of living *Acari* wallowed and tumbled about." The cases in which such a severe form of *scabies* has occurred have been characterized by extreme filth and neglect of treatment, and the irritation and course of the disease has tended to a fatal termination by pneumonia and hyperæmia of the brain.

**Treatment.**—Such applications are to be made to the skin that, while they tend to kill the *Acari*, they will not increase the irritation of the dermis; and if the *Acari* are thus destroyed, the eruptions will in general subside in due course.

If much irritation of the skin prevails, warm baths are to be prescribed, and opium may be given internally.

In healthy adults the whole body of the patient ought first of all to be thoroughly scrubbed over with good black (soft) soap, and the process continued for at least half an hour. The patient should then get into a warm bath, in which he should remain for another half hour. Having thus washed and dried himself thoroughly, he is to rub himself over with the following ointment:

R. Subcarbonatis Potassæ, ʒj; Sulphuris, ʒij; Axungiæ, ʒxij. Misce.

[The ointment used at the St. Louis Hospital, Paris, by Dr. Hardy—who revived the method introduced by Bourdin (1812) and materially abridged the time employed—is composed of sulphur ointment, to which subcarbonate of potash is added in the proportion of half a drachm to the ounce; 3 to 4 oz. of ointment, and 1 lb. of soap, are used to one patient. The ointment is made more certain by adding ʒj oleum anisi.]

Next morning a warm bath is to be taken, to clean the surface of the body from the remains of the anointing of the previous night. The cure ought now to be complete, so far as the destruction of the *Acari* are concerned (HARDY, HELMERICH, ANDERSON). The genuine pomade of Helmerich is one-third stronger than that which has been just quoted from Dr. Anderson, who considers it too irritant. The potash in the black soap and ointment acts as a solvent of the epidermis, and thus allows the sulphur to come into more immediate contact with the *Acarus*. A warm bath and plenty of hot water ablution completes the cleansing process.

The treatment may, however, be inapplicable to children, females, and men with delicate skins, or constitutional affections of the skin.

In them, although the principle of treatment is the same, the process of cure must be more slowly conducted by less powerfully

irritant substances. The patient having cleansed himself thoroughly in a warm bath, with ordinary yellow soap, the following lotion may be applied:

R. Calcis, 3ss.; Sulphuris, 3j; Aquæ, 3viiij. These ingredients are to be boiled and stirred constantly till a homogeneous mixture is produced, which is to be strained through a sieve. [The fluid is to be decanted and kept in a well-stoppered bottle.]

These ingredients ought to produce a quantity more than sufficient for one person, and should be rubbed into the skin, not too roughly, [for half an hour], every night for several evenings. The cases of scabies in the Belgian army are treated by this lotion, (VELMINSKZ, ANDERSON), [KENDALL, NICHOLLS].

When the person affected is predisposed to *eczematous* eruptions, the following application is recommended:

R. Sulphuris, Olei Fagi, āā 3vj; Saponis Viridis, Axungiae, āā lbj; Cretæ, 3iv. Misce.

This ointment should be well rubbed in, after the skin has been prepared for it by the use of the warm bath and cleaning the body with common yellow soap. The potash in the black soap of the ointment acts as already stated, the chalk tends to remove the epidermis mechanically, the tar counteracts the tendency to eczema, and the sulphur destroys the *Acaris*. The ointment ought to be left on overnight (if the skin is not too irritable), and should be washed off in the morning (WILKINSON, HEBRA, ANDERSON). Specific printed directions should be given to each patient; and cards are useful for this purpose, similar to those in use in the Dispensary for Skin Diseases in Glasgow. The following are the directions *printed* on each card, and which is given to each patient along with the quantity of ointment required:

"1st. Scrub the *whole* of your body (except the head) as firmly as possible, without hurting yourself, with black soap and water.

"2d. Sit in a hot bath for twenty minutes, or if you cannot get a bath, wash yourself with hot water thoroughly.

"3d. Rub some of the ointment thoroughly into the skin of the whole body (except the head) for twenty minutes. Let the ointment remain on the body all night.

"Repeat these processes every night for three nights, and then return to the dispensary.

"Besides, put all your washing clothes into boiling water, and iron all your other clothes *thoroughly* with a hot iron."

If such methods are systematically carried out, itch cases ought never to occupy hospital beds, either in civil or in military life.

The ordinary compound sulphur ointment of the pharmacopœia is also an efficient remedy.

[Dr. Pastau, of Breslau, has recommended *liquid storax* as the most certain of antipsorics, and it is said to have been successfully adopted in



not communicable to animals and to man? It is known how intimately the diseases of man and animals are related with the occurrence of famines and the prevalence of unsound or unwholesome food, and of famines with the diseases of vegetable and animal life, as much as with the destruction and loss of food. The black sporules of *Ustilago hypodytes*, which cause disease in grasses in France (the grass smut), and those of *Ustilago vittata*, which cause similar disease in the grasses of India, are known to produce most injurious effects upon the haymakers in the former country (LEVEILLE). In places favorable to the multiplication of *fungi* they often commit extensive ravages. Among the silk-worms in the silk manufactories of Italy, *fungi* are the cause of more extensive destruction of such animals than we have any correct idea of. Under the names of "mildew," "blight," "smut," "brand," and the like, *fungi* commit extensive damage among living plants, as the farmer and orchardist know too well. It has been asserted that *fungi* are uncommon in tropical countries; but it is doubtful if this is true; and the *fungus disease of the foot in India*, so well described by the two Carters, show that *fungi* are capable of giving rise to a disease almost dangerous to life in that country. It therefore behooves the pathologist to study carefully the nature of those diseases in animals and vegetables, as well as in man, whenever he has an opportunity, and especially in India. It is not in all cases easy to determine whether they are the cause of morbid states, or whether, as some think more likely, the diseased tissue has merely afforded a suitable nidus for their development. It is certain that wherever the normal chemical processes of nutrition are impaired, and the incessant changes between solids and fluids slacken, then, if the part can furnish a proper soil, the cryptogamic parasites will appear. The soil they select is for the most part composed of epithelium or cuticle, acid mucus or exudation. Acidity, however, though favorable for their growth, is not indispensable; since some of the vegetable parasites grow upon alkaline or neutral ground, as on the ulcerations of the trachea, or in fluid in the ventricles of the brain. Certain atmospheric conditions seem favorable to the occurrence of those vegetable parasites. For example, *Tinea tonsurans* may be quite absent for years in places such as workhouses, where it commonly exists, and then for several months every second or third child in the place gets the disease. It has been observed that some of these parasitic diseases can be propagated by transference of the plant, as in various forms of *tinea*, and that the disease can be cured with the greatest readiness by the chemical agents which are most destructive to vegetable life.

These vegetable parasites have been shown to be capable of transmission from animals to man. It has been recently proved by Devergie that *T. tonsurans*, for example, is transmissible by contagion from horses and oxen to man, and that the parasite has given rise either to the same form of *tinea* disease or to another; but he ascribes both species of parasitic disease to the growth of one and the same parasite—namely, the *Trichophyton*. Von Bärensprung, of Berlin, bears similar evidence. He rubbed on his forearm some of



sary with favus of the non-hairy parts of the body. The father and two other children were similarly affected. Mice previous to this had abounded in the house, and a cat was got to kill them. On examining the cat numerous favus cups were detected on the tops of its forepaws. (3.) An eruption was noticed on a little girl, and soon afterwards a sister, the mother, the baby, and a work-girl were similarly affected. The disease proved to be favus. Five weeks previously a number of mice had been caught in the house, and which had been much handled by the children. Several mice in the house were then caught and examined, and on the back of one of them, near the tail, a characteristic favus cup was seen, while the sides of the ears and head of another were eaten away by the disease. On the crusts being examined with the microscope, the *Achorion* was detected in great abundance.\*]

On the other hand, it is shown that animals may contract parasitic diseases of the skin from human beings similarly diseased. Dr. Fox mentions an instance of a white cat, a great pet with the children of a family of nine, which contracted the *mange* and *T. tarsi* from *T. tonsurans* affecting five of the children. The fungus of the *mange* in the cat is the same fungus as that of *Tinea* in man—namely, the *Trichophyton*.

[Köbner succeeded in producing favus in rabbits, by inoculating them with the *Achorion* taken from the human subject.†]

The principal vegetable parasites associated in man with special morbid states have been enumerated as follows:

1. The *Trichophyton tonsurans* vel *Achorion Lebertii* (ROBIN), which is present in the three varieties of *Tinea tonsurans*—namely, *T. circinatus* (ringworm of the body), *T. tonsurans* (ringworm of the scalp), and *T. sycosis menti* (ringworm of the beard).

2. The *Trichophyton sporuloides* (VON WALTHER), together with the above, which are present in the disease known as *Plica* vel *Tinea polonica*.

3. The *Achorion Schönleinii* (REMAK) and the *Puccinia favi* (ARDSTEN), which are present in *Tinea favosa* (the honeycomb ringworm).

4. The *Microsporon mentagrophytes* (GRUBY), which is present in *Sycosis* or *Mentagra*.

5. The *Microsporon furfur* (EICHSTADT), which occurs in *Pityriasis* vel *Tinea versicolor*.

6. The *Microsporon Audouini* (GRUBY), which is present in *Porriago* vel *Tinea decalvans* (*Alopecia areata*).

7. The *Mycetoma* vel *Chionyphe Carteri* (H. V. CARTER, BERKELEY), which gives rise to the disease known as “the fungus foot of India,”—a cotton fungus occurring in the deep tissues and bones of the hands and feet.

8. *Oidium albicans*, or “thrush fungus” (ROBIN) of diphtheritis and aphtha.

\* [On the Non-identity of the Parasites met with in Favus, Tinea tonsurans, and Pityriasis versicolor. By Dr. McCALL ANDERSON. *British and Foreign Med.-Chir. Review*, vol. xxxviii, 1866.]

† [Klinische und Experimentelle Mittheilungen aus der Dermatologie und Siphilologie, von Dr. Heinrich Köbner. Erlangen, 1864.]

9. *Cryptococcus Cerevisæ* (KUTZING), *Torula Cerevisiæ* (TURPIN), yeast plant in bladder, stomach, &c.

10. *Sarcina ventriculi* (GOODSIR), or *Merismopædia ventriculi* (ROBIN), in the stomach.

It would have been better if these *fungi* had been described in the first instance without specific names. The fact of specific names having been assigned to each of them has drawn attention from the important part which these *fungi* perform in the work of decomposition. They may be forced to fructify by placing them in a globule of water surrounded by air, and placing them in a closed cell; and until the fungus has thus come to maturity it is worse than useless to give them names—so many different forms in an undeveloped state being all capable of reference to one common mould (BERKELEY).

The plants forming on mucous membranes, or in the contents of cavities lined by mucous or serous membrane, are in most cases only of secondary formations, and their exact pathological significance is unknown.

The pathognomonic sign of all the parasitic lesions of the surface in man and animals is the infiltration or destruction of hairs (*tineæ*) and epithelial textures (*muguet*, *thrush*, *öidium*) by the *sporules* of a fungus, and which, by union or by growth, form elongated branches, or *mycelium*.\*

The diagnosis of such *fungi* on the skin, hair, or epithelium, can only be effected by a careful and skilful microscopic examination; and it is always absolutely necessary to use the *liquor potassæ* in the examination of all *tonsurant* appearances of the hair, of all *idiopathic* bald patches, and of all brown or yellow-colored scurfs, for *sporules* are frequently detected which had escaped observation before *liquor potassæ* had been used (BAZIN, FOX, ANDERSON). Sufficient time must also be allowed for the parts to become transparent under the action of this reagent.

The parasitic lesions of the skin are, as a rule, unsymmetrical, and hence they differ materially in this respect from syphilitic cases. They differ also no less essentially from the eruptive diseases of the skin. An eruption is no necessary part of these parasitic lesions; but, from the irritation established in the true skin, eruptions of various kinds may occur. Eruption thus often precedes the detection of a fungus, and, as a rule, very often follows its existence; and they who dispense with the microscope in the diagnosis of skin affections cannot avoid confounding severe eruptive with parasitic lesions, because they disregard the pathognomonic evidence of such lesions already indicated (FOX). The term *herpes*, therefore, as applied to these parasitic affections, is an objectionable term; because it has been already used to indicate a vesicular eruption, namely, *herpes zoster*, which invariably exhibits large typical vesicles.

There seems to be a peculiar condition of nutrition best fitted for parasitic growths of a vegetable nature, just as some constitu-

---

\* These *fungi* have sometimes been popularly but erroneously termed *conferveæ*. The *conferveæ* belong to the *algæ*, and for the most part grow in salt or fresh water.



tions and states of the system are best suited for the propagation and development of entozoa; but the exact circumstances which predispose to the growth of these *entophytic fungi* upon the human body are not better known than those which predispose the body to receive and develop certain morbid poisons of a specific kind, known to multiply during the course of the disease, and to throw off material capable of propagating and spreading the same kind of disease. With regard to the parasitic diseases of the skin and hair, a failure of the vital powers to carry on the healthy processes of life seems ordinarily to be one of the inviting causes of such a development of true *fungi* as would constitute a disease. A special nidus or soil is necessary, just as a predisposition is necessary in the case of the spread of miasmatic diseases; yet care must be taken in both instances not to confound the co-operating cause with the special or peculiar poison or germ. Both Robin and Bazin recognize a condition of the hairs (dependent, perhaps, on constitutional causes) which appears to be essential for the growth of the plant; for sometimes the disease disappears spontaneously, and the fungus dies without treatment.

[Malnutrition and feeble health of the subject seem to favor the development of both vegetable and animal parasites,\* and in scrofulous persons they are common and tenacious; but no condition is so fitting as uncleanness, the fungus there finding a good soil where it may rest unharmed, with no let to its growth, and this point is particularly insisted upon by Hebra. They affect particularly the skin of children.]

The evidence is now very strong which points to the various *fungi* already mentioned as mere varieties of two or more species (HEBRA, LOWE, HOGG, FOX, BERKELEY).

There are numerous facts which justify the belief that there exists but one essential fungus, whose sporules find a soil for development and growth upon the surface, or even within more secluded portions of the human body; and that varieties in the growth of that fungus are due to differences in the constitution of the individual, to the moisture, exudation, soil, or temperature under which the development of the fungus takes place. The exact nature of these differences is not yet understood; but the production of irritant acids and gases is a constant accompaniment of the growth of such parasites, by the chemical action of the vegetable cell; for it does not undergo development without exciting a chemical decomposition in the pabulum on which it feeds; and the different stages in its growth give rise to alcoholic, acid, and putrefactive fermentation. Of the latter there is ample evidence in many of these parasitic skin diseases, and especially in favus, the odor of which closely resembles that of some *methyamine* compound (LOWE). Thus their irritant action very soon may establish an eruption. But the ratio

---

\* [Cl. Bernard states that frogs are subject to parasitic fungi, which in time cause their death; and that if a healthy frog is put into a jar with others so diseased, he will, for awhile at least, resist contagion; but if one which has been for some time in captivity, and whose nutrition is impaired, be thus placed, he becomes at once covered with fungous growths.]





phere, ready to rest and grow upon any suitable pabulum. Remak made like experiments, but was unable to reach any positive conclusion; while Köbner put the question to the practical test of repeatedly inoculating himself, Strube, and others with the *Penicillium glaucum*, without any effect. "Now," as Dr. Anderson very fairly remarks, "if the *Penicillium glaucum* were identical with the parasites of favus, ringworm, and Pityriasis versicolor, one would naturally have expected that he would have been as successful with it as he was in his inoculations with the *Achorion Schönleinii*, the *Trichophyton*, and the *Microsporon furfur*;" and he thinks, "it must be conceded that further proof is required, before we can admit that the parasites productive of favus, *Tinea tonsurans*, and Pityriasis versicolor are identical with the *Aspergillus glaucus*" (ANDERSON, *loc. cit.*.)]

Peculiarities have also been observed in the growth of the fungus, which may be explained as due to the stage of development and conditions of growth at the time it was examined (*Path. Society Trans.*, vol. vii, p. 395). The same *fungi* during their growth are known to assume very different forms and appearances. It thus happens that the same species has not only been described under different specific names, but even referred to different genera. Fries states that he has traced no fewer than eight genera of different authors to mere degenerations or imperfect states of one particular fungus (*Thelephora sulphurea*); and Nees von Esenbeck states that the same fungoid matter which develops a certain fungus in winter (the *Sclerotium mycetospora*) will develop another fungus in summer (the *Agaricus volvaceus*.) Professor Henslow showed that some of the supposed species of *Uredo* are forms of *Puccinia*, *Aegma*, and the like.

The identity of these *fungi* associated with skin disease has been likewise proved by clinical observation. In patients affected with ringworm of the head (*T. tonsurans*), patches of ringworm of the body (*T. circinatus*) are frequently seen on other parts of the skin, more especially on the neck, where the patches of the skin and scalp are often continuous (JENNER, ANDERSON). Instances of the converse of these observations are recorded by Dr. Fox, where the disease from the skin extended upwards to the scalp, with the characters of *T. circinatus* on the skin and *T. tonsurans* on the scalp (*Lancet*, Sept. 17, 1859). Hutchinson states that once, in examining with the microscope the parasite from a case of *T. tonsurans*, he inoculated himself by mistake, and there resulted a well-defined patch of *T. circinatus* on one side of the neck (*Med. Times and Gazette*, Jan. 12, 1861). Dr. Anderson mentions that in cases of *sycosis* he has more than once found the external aspect or back of the hand or wrist the seat of *T. circinatus*, owing to the patient rubbing the itchy and diseased portions of the chin with these parts, thus giving an opportunity for the transmission of the parasite (*Parasitic Affections of the Skin*, p. 50). The ringworm of the skin, he also observed, might give rise to *sycosis*. Dr. Fox relates that while examining a patient with *T. circinatus* he had two or three vesicles of *simple herpes* just appearing at the time on his own lip. The fungus became implanted amongst these vesicles; and the herpetic eruption

me irritable, inflamed, and pustular; were actually split up by the fungus down to the follicle.

*Sycosis* is also shown to be produced from the same fungus. Fox has shown that *favus* and *sycosis* are different manifestations of the yeast plant; and *Trichophyton* have been seen in *T. tonsurans* may be produced from the invasion of the "thrush" (Fox). The nail fungus, or an *Oidium* (KUCHENMIST), while the ear fungus is an *Aspergillus* used in illustration of the text (show the sameness in the form of the diseases; and if contrasted with Fox's admirable work on *The Principles of Dermatology* showing the sporules and mycelium of the fungi, and with Figs. 86, 87, and 88, cannot fail to strike the observer that the fungi peculiar to the diseases about the head are identical. Nevertheless, the lesions induced necessitate the description of separate diseases, for the treatment varies with each disease; and the clinical aspect of each is determined by the stage of development, growth, and duration.

The most eminent dermatologists are now of the opinion that the parasitic affections of the skin are not essentially the presence of fungi in these diseases, but are described as such to be degenerations of the skin. (2.) Some admit that they are accidental and not essential formations. (3.) Others believe that parasitic fungi may be generated spontaneously, or may be introduced from without where they are found. (4.) The camp of those who believe that several fungi produce the parasitic affections of the skin maintain that they are due to the presence of the fungi. A recent contribution to the *British and Foreign Medical Review* (July, 1866), Dr. McCALL ANDERSON, who has been occupied with great care, and who, in his *Affections of the Skin*, was the first to recognize the correctness of Bazin's opinion—that *Tinea* and *Tinea sycosis*, are all due to the presence of the *Trichophyton*—has presented very convincing evidence. *Trichophyton* (the parasite of the three diseases), *Schönleinii* (the parasite of scald head), and *trichophyton* (the parasite of chloasma, Pityriasis, and other growths. His proofs of the non-identity of the fungi are from: (1.) The results of inoculation. (2.) Microscopic examination. The following are brought forward in support of his probabilities of successful inoculation with the *Trichophyton furfur*, the same parasitic disease.

that from which the parasite was taken. (2.) Of the innumerable cases occurring in the human subject, illustrative of the contagious nature of favus, *Tinea tonsurans*, and Pityriasis versicolor (chloasma) which have been recorded, there is no authentic case in which one of these diseases gave rise to one of the others. In one of Hebra's plates there is an instance of ringworm and favus on the same person at one time. This is no doubt very rare. Dr. Anderson says, he has never met with a case, and that amongst the 1300 cases of parasitic affections of the skin treated at the Dispensary for Skin Diseases, Glasgow, during the last four years, there were numerous examples of the contagious nature of tinea, favus, and chloasma, but there was not a single instance of one of these diseases giving rise, by contagion, to one of the others. He remarks, that when the coincidence happens, it is no more constitutive proof of the identity of these diseases than do instances of the coexistence of psoriasis and ringworm show the identity of these two affections. (3.) The difference in the appearance of favus, *Tinea tonsurans*, and Pityriasis versicolor (chloasma), when fully developed, is so very striking, as to lead to the belief that they are produced by separate parasites. (4.) There is no authentic instance on record of the transition of one of these diseases into one of the others. (5.) The difference in the appearance of the Achorion, *Trichophyton*, and *Microsporon furfur* is sufficiently striking to enable the observer in many cases to form a correct diagnosis from the microscopic examination alone.\* (6.) Of the numerous instances on record of the transmission of favus and *Tinea tonsurans* from the lower animals by contagion or inoculation, favus has always given rise to favus, and *Tinea tonsurans* to *Tinea tonsurans*.]

#### RINGWORM—Syn., *TINEA TONSURANS*.

LATIN Eq., *Tinea tonsurans*; FRENCH Eq., *Tinea tonsurans*; GERMAN Eq., *Tinea tonsurans*—Syn., *Ringwurm*; ITALIAN Eq., *Tinea tonsurans*.

**Definition.**—An affection implicating the hairs of the skin, scalp, or chin, and usually assuming a circular form. The hairs become dry and brittle, having a tendency to crack or break across. Itching accompanies the primary eruption, which is generally at first erythematous, with slight swelling, and a fungus ultimately appears (*Trichophyton tonsurans*), which had been developing between the epidermis and the true skin. The fungus has a pure white color and powdery aspect. It covers the epidermis between the hairs, and forms around them a complete white sheath. Inflammation of the hair follicles and of the surrounding tissues occurs; and when pus forms, the fungus is destroyed at the expense of obliteration of the roots of the hair, when perfect baldness ensues (BAZIN, ANDERSON).

**Pathology.**—The nature of this disease is to be studied in the botany of the cryptogamic parasite called the *Trichophyton*, discovered by Malmsten in 1845. It consists of oval transparent spores

---

\* [The spores of the *Achorion* are, on an average, about 8000th of an inch in diameter, and many of them are oval; those of the *Trichophyton*, are much smaller, being, on an average, about the 7000th of an inch in diameter; while the spores of the *Microsporon furfur*, although nearly as large as those of the *Achorion*, are more uniformly rounded, and have a remarkable and characteristic tendency to run together, and form clusters, like bunches of grapes (ANDERSON).]



face affected is large, the circle is apt to become incomplete, so that various segments of circles appear. The disease may terminate spontaneously, the parasitic fungus being very superficial, the hairs small and rudimentary, so that the parasite dies for want of nourishment. It is apt to affect the face, the neck, the back, and outside of the wrist and hand (BAZIN, ANDERSON).

2. *Ringworm of the Beard* (*Tinea sycosis*) is met with on the upper lip and hairy parts of the cheeks, as well as on the chin, when it affects men; but the hairs of the axillæ or genital organs of females are not exempt from this disease. It commences exactly like *T. circinatus*; but it is not till the deeper structures are involved, and when small indurations occur, surmounted by pustules resembling *acne*, and when the hairs can be pulled out with ease, that the attention of the patient is attracted to the affection. The hairs are thickened, the bulbs flattened and more or less disorganized. The longitudinal fibres of the hair are separated by masses of sporules embedded between them; and where the fungus accumulates, nodes on the hair indicate the site of such accumulation. The medullary part of the hair is quite disorganized (Figs. 46 and 47), and may disappear altogether. In the advanced stage of *sycosis*, when inflammatory and suppurative phenomena prevail, the fungus is difficult to find.

3. *Ringworm of the Scalp* (*Tinea tonsurans*) is for the most part met with in children. It generally makes its appearance first in the form of rounded patches on different parts of the head, of a scaly or pityriasis-like inflammation. From the irritation induced small vesicles may form. The hairs in the first instance are dull, dry, twisted, and easily extracted; but as the disease advances they become very brittle, and break on attempting to extract them; and as they become more and more friable they break of themselves within a line or two of the skin (BAZIN, ANDERSON). The twisting of the hairs, so frequently observed, is due to—(1.) Plugging up of the follicular orifice by secretion, and detention of the upper part of the shaft of the hair, while its growth at the papilla still continues. Half an inch in length of hair may sometimes be pulled from beneath the false operculum. (2.) It may be due to the presence of *mycelium* in the follicle, clinging to the hair on one side and to the follicle on the other. It thus blocks up the follicle, and holds the hair (while still growing) to the diseased spot (Fox). The epidermis and stumps of the hair become covered over with a characteristic grayish-white powder, which ensheathes the hair. This powder consists of the *sporules* of the fungus. There is slight elevation and puffiness of the skin of the diseased parts, while its color is bluish or slate-colored in dark subjects, and grayish-red or yellow in fair persons (BAZIN, ANDERSON). This elevation and puffiness of the skin is due to a granular layer or *stroma*, which, on the addition of *liquor potassæ*, is seen under the microscope to be due to *sporules* of the fungus closely packed together. The amount of fluid influences materially the size of the *sporules* (ROBIN, Fox). When pustules and yellow crusts form, the detection of the fungus is more difficult.



is cured. It is first of all necessary to remove the hair. This is in part generally accomplished before the case comes under treatment, by the course of the disease; if it has not been sufficiently done, "epilation" can be accomplished by a chemical agent, or by extraction with pincers (Fig. 48). The forceps most suitable for this little operation are those about three inches long, having a weak spring, so that the hand may not be fatigued in using them. They should be made so that the two extremities come together very exactly, and do not slide the one upon the other. Each extremity should be a couple of lines broad, so that a fasciculus of hair may be caught up at one time when required; and should be furnished on the inside with very fine, but at the same time blunt, transverse denticulations, so that they may not cut across the brittle hairs. M. Bazin recommends an ointment composed of lime and carbonate of soda, of each one part, and thirty parts of lard, as an agent to remove the hair. The *oil of cade*, however, appears to be the best depilatory known, and with this mode of treatment epilation with the pincers may be combined. If the hairs are pulled out in the proper direction, there is very little pain, especially after the sensibility of the skin has been blunted by the use of the *oil of cade*. [After epilation, a lukewarm local douche should be used for ten or fifteen minutes.] The removal of the hairs permits a "parasiticide" solution to be applied to the hair follicles, within which are the prolific spores of the fungus. For this purpose M. Bazin recommends either a solution of *bichloride of mercury* (1 part to 250 of water) or an ointment of the *acetate of copper* (1 part to 500 of lard), about two grains to an ounce of water; and a little *alcohol* or *muriate of ammonia* should be used to facilitate the solution of the mineral. The *oil of cade* should be mixed with *glycerine* in the proportion of half a drachm to a drachm of the oil to an ounce of *glycerine*. Kuchenmeister's experience shows that the alcoholic solutions act most powerfully.

FIG. 48.\*

Dr. Parkes has used, with excellent effect, a solution of the *pernitrate of mercury*, about one part to thirty or forty of water. This is, however, a very powerful remedy, and is to be cautiously used, as it easily blisters the scalp; also an ointment composed of sulphate of copper (one part), alum (three parts), and lard (twenty to thirty parts, according to the age of the patient). Probably, however, a better parasiticide than any of these is the *sulphurous acid* [applied pure by means of a glass rod, or a piece of lint may be dipped in a saturated solution of the acid, and kept on the affected parts, with oiled silk over it, to prevent evapora-

\* Forceps for epilation (Dr. ANDERSON).





the surface of the skin, forming the yellow honeycomb-like masses which gave the specific name *favus* to the disease, and which, from their frequent buckler-like shape, suggested the term "scutulata." A cuticular elevation is seen, beneath which is a small favus. When the cuticle is raised, a drop of pus sometimes issues; hence the error of those who have considered this disease always pustular. Generally, however, there is no pus or liquid of any kind: the plant grows, and the cuticle over it (supposing it has not been forcibly detached) finally separates, leaving the favus exposed to the air. A third form of the disease is that in which the fungus attacks the nails, and occurs for the most part in those who have been long affected with the favus of the hair follicles, the fungus taking root and germinating beneath the nail (Fig. 49). After the spores have commenced to germinate between the superficial and deep epidermic layers, the nail becomes thickened over the affected part, and its color becomes gradually more and more yellow, owing to the favus matter shining through it. As the fungus increases in growth, it gradually presses on the nail, rendering its longitudinal striæ very evident, and ultimately leading to the formation of fissures in it. As the pressure of the nail increases, its substance gets thinner and thinner, till perforation occurs; and then a favus cup makes its appearance externally, but more or less deformed (ANDERSON).

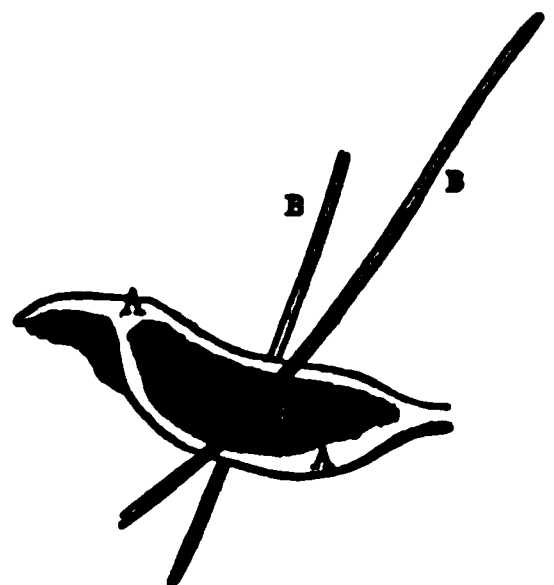
It is important to notice that at first there is, at the point where the favus is about to form, only an increased secretion of epidermis; and sometimes the under surface of the favus is coated by cuticle, which separates it from the compressed and attenuated derma. As it increases in size, and becomes more prominent, the epidermic covering is ruptured. Each favus crust is also enveloped in a capsule of amorphous structure, within which is inclosed the true favus matter (Fig. 50).

The favus consists of the *mycelium*, the *spores*, and the *receptacles* of the *Achorion*, together with a finely granular amorphous layer, which forms the external coat of the favus, and is the representative of the amorphous "stroma" which often accompanies the mycelium of *fungi*. In the favus another and distinct fungus can sometimes be found—namely, the *Puccinia favi*—which is easily recognized: it has one extremity (the body) rounded and composed of two cells

FIG. 49.\*



FIG. 50.†



\* (A, A.) Upper surface of nail; (B, B.) Lower surface of nail; (C, C.) *Favus* matter (white in the wood-cut, yellow in the original), running upwards and forwards between the laminae of the nail (ANDERSON).

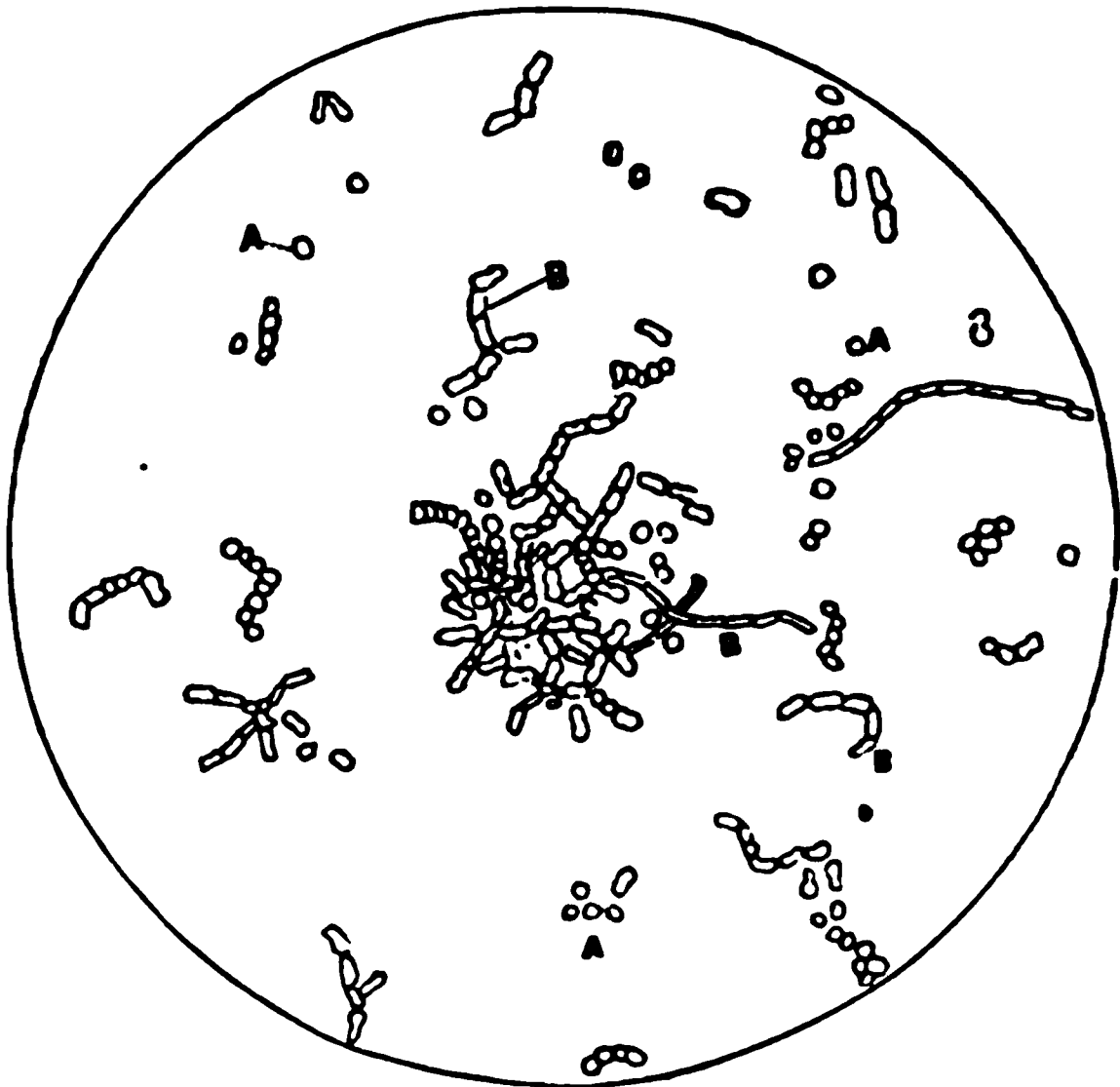
† Favus cup.—(A, A.) Amorphous envelope; (C, C.) Favus matter; (B, B.) Hairs traversing the Favus cup (ROBIN).

of unequal size, a superior and an inferior; the other extremity is prolonged into a jointed stem or trunk.

When a little of the favus matter is broken up and examined microscopically, after being acted upon by solution of potash, it is seen to consist of numerous little oval or rounded bodies, the sporules of the fungus having a diameter of about  $\frac{1}{800}$ th part of an inch (Fig. 51).

A number of cells united end to end form simple or jointed and branching tubes (Figs. 52 and 53), developed from the sporules (Fig. 51). Little granules or nuclei may be seen in the interior of

FIG. 51.\*



the spores. The tubes vary in diameter, and hairs in the vicinity of the favus crusts are impregnated with the fungus. The disease has been communicated by inoculation from man to man, and from mice to cats, and thence to man (BAZIN, DRAPER, FOX, ANDERSON).

**Symptoms.**—Favus is the most common and the most inveterate form of scald head. The disease commences with a slight pruritus or itching of a few hours' duration, followed by an eruption of small red *vari*, sensible to the touch and to the sight. These augment in size, and, before twelve hours have passed, a yellowish point forms on each of their apices, at first so small as to be only visible under a glass of considerable power. The surface appears now as if covered with specks of a sulphur-yellow color, and each *varus* appears as if set in the skin, with an umbilicated or de-

---

\* Fungus matter from a favus crust.—(A, A, A.) The isolated sporules; (B, B, B.) Chains of sporules (Dr. ANDERSON).

pressed centre. If any fluid exudation exist, it does not remain so, but concretes into a dry, brittle, candied, honeycombed-looking

FIG. 52.\*

  
1/100th of an inch  $\times$  340 diameters (Dr. ANDERSON).

scab or crust, which retains the form of the pustule, is similarly cupped or depressed in the centre, covered by the epidermis, while

FIG. 53.†

---

\* Fungus matter from a favus crust, showing branching tubes running inwards to the centre of the figure from the epithelial scabs and sporules at the edges (ANDERSON).

† Showing the mode of reproduction of the *Achorion*, or fungus of the favus (after BENNETT).



The *Porrigo lupinosa* and *Porrigo scutulata* are accidental varieties, in which the scab resembles a lupine, rather than the cell of the honeycomb, and is very rarely seen; or the appearance of the scab is shield-like; and when of some extent and well marked, the patch is soft, doughy, and painful when pressed upon. Some of the hair appears to be removed by the roots, while other portions are broken off near the scalp, the roots remaining. Those which remain are readily removed by friction, and if pulled, have scarcely any hold of the scalp.

**Treatment.**—The treatment of the various forms of *favus* is now very strictly determined. Some practitioners, however, still rely entirely on a constitutional treatment, such as small doses of rhubarb and soda, small doses of mercury, some preparation of iron; or on vegetable tonics, as the infusion of cascarilla or compound infusion of gentian. With such treatment, if the health improves, it is believed the fungus will spontaneously disappear. Others, again, as entirely rely on a local treatment, attempting to exterminate the disease by cauterization, or by applying some favorite ointment; and the catalogue of ointments used for this purpose includes all that have at any time been admitted into the pharmacopœia.

As in the last-described disease, the cryptogamous parasite must be destroyed, and its germs eradicated.

The best method to accomplish this, is, in the first instance, to shave the head and apply a poultice till all the scabs, or nearly so, are removed; and this being effected, the whole hairy scalp, or site of the *favus* fungus, should be anointed with some of the following applications: The *tar ointment* (*ungt. picis liquidæ*) has hitherto been the orthodox application. This ointment should be washed off night and morning with soft soap and water, and be as often reapplied. The head should be shaved twice or thrice a week, and where there are other children, the affected child should be isolated as much as possible, to prevent the disease from spreading. This form of *porrigo*, in the early stages, will sometimes yield by washing the part with the *oleum terebinthinæ* night and morning, and cutting the hair close.

The *Favus scutulata* is a disease often rebellious to every mode of treatment, but, applied at a favorable moment, a simple method may succeed. Dr. Willis has seen the disease yield to fomentations, or to bread poultices. The application of the lunar caustic round the patches, about a line from their outer margin, is another favorite method of treatment. In the latter periods of the disease, Dr. Willis recommends—

A solution of *Sulphate of Copper*, in the proportion of seven grains to ten ounces of water; or of the *Nitrate of Silver* in the same proportions. The mild ointment of the *Nitrate of Mercury*, a salve of the black sulphuret of the same metal (*Sulphuretum Hydrargyri Nigr.*, ʒj ad ʒij, Adipis ʒj); the *Unguentum Picis*, an unguent of the *Cocculus Indicus* (ʒj to ʒij, Adipis ʒj), may be tried one after the other; and in different instances each will have the merit of the cure.

[Hebra, following the recommendation of Kuchenmeister, has succeeded with, first rubbing the parts with lint soaked in a lotion of *Vera-*



In the early stage of the disease the hairs appear dull and lustreless, and more easily extracted than healthy hairs. The skin is reddened, swollen, and slightly itchy. A whitish matter may be seen on the diseased skin and hairs, which is due to the sporules of the fungus. The hairs suddenly fall off from the affected parts, and a round bald patch is left, which is perfectly white, contrasting in its whiteness with the parts of the scalp or skin provided with hairs. The fungus may also be developed in the nail, like favus (BAZIN).

FIG. 55.\*

There is an affection which should be distinguished from the *Porriago decalvans* (or *Alopecia circumscripta*), and which is characterized by a rapid disappearance of pigment from both skin and hair, with or without alopecia. M. Bazin includes it in his *Tinea achromatosa* (*Teigne achromateuse*), but does not mention the fact that alopecia is not constant; and states that a parasitic plant is present. It is probable, however, that something more than a fungus exists, to cause the total disappearance of pigment from a considerable portion of dermis. Besides, when the hairs return, they are at first quite white and downy, like those on children, and only gradually regain color; whereas, if the vitiligo were owing to a plant, they would most likely not grow at all. The disease appears to be allied to those obscure pigmentary changes which have a much deeper seat than the surface of the body (PARKES). Vitiligo is sometimes a congenital affection, and seems to consist in an abnormal distribution of the pigment of the skin; so that there are irregular patches which are quite white, and altogether wanting in pigment, but are surrounded by skin provided with an excess of coloring matter.

H

\* Fungus of the hairs resulting in *Alopecia* — (A, F.) Lower part of the hair; (F, G.) Root of the hair without the capsule; (C.) Spheroidal swelling of the hair, due to the accumulation of sporules; (X.) Between the longitudinal fibres of the hair; (D.) Rupture of the longitudinal fibres; (I.) Sporules and tubes of the parasite; (H.) A group of sporules proceeding from G, the ruptured root (ANDERSON).





shade almost black. The color has been said to resemble diluted bile.

A microscopic fungus, to which the name of *Microsporon furfur* was given by Robin, is the essential cause of the disease. It was discovered by Eichstädt in 1846. Soon afterwards it was described by Snyter and by Sprengler, who gave a drawing of it. On putting a little of the dust from the desquamating surface under the field of the microscope, and adding a drop of *liquor potassæ*, scales of epidermis are seen mingled with the sporules and tubules of the fungus. The sporules are oval or rounded, and usually collected into large clusters like bunches of grapes, and are so characteristic as almost to be pathognomonic (ANDERSON). The tubes are short and branching.

FIG. 56.\*



Dr. Anderson and Mr. Startin give numerous instances which prove that chloasma is a disease capable of being propagated from one person to another. It is a common affection with scrofulous persons especially, and may not unlikely be favored by wearing the same flannel day and night, neglecting to wash the body for fear of catching cold (ANDERSON). It is not uncommon for such people to wear the same flannel next to the skin for a week, a fortnight, three weeks, and among the poor even for a month. And it is by no means an uncommon thing for them to wear the same flannel night and day, not once removing it from the moment it is put on till the time it is considered desirable to have it washed. The consequences of such habits are an accumulation on the surface of the skin of its secretion, and of undetached epithelium, and the consequent formation of a nidus favorable to the growth of the *Microsporon furfurans* (Dr. Jenner, *Med. Times and Gazette*, 1857, p. 651).

**Treatment.**—Local applications constitute the principal part of the treatment. A solution of bichloride of mercury, in the proportion of two grains to an ounce of water, applied over the affected parts once or twice daily, is generally effectual in destroying the progress of the fungus. Mercurial or sulphur baths have a similar effect, either singly or combined, care being taken to avoid salivation. The use of black soap night and morning is recommended by Dr. Anderson, or the use of the following mixture:

\* Shows the grape-like arrangement of the sporules and the short branching tubes of the *Microsporon furfur* in chloasma (after Dr. ANDERSON).



The fungus disease and material of the fungus assumes various forms, three of which may be considered typical:

1. The first form is that in which the bones of the foot and the lower ends of the leg-bones, just above the ankle (for the disease never ascends higher), are perforated in every direction with roundish cavities, varying in size from that of a pea to that of a nut or pistol-bullet (Fig. 57), the cavities being filled up with a dense fungous mass, of a sienna red within, but externally black, and resembling a small dark surface, from which a purulent fetid discharge is poured out, often accompanied by little pieces of the fungus. The masses and granules are embedded in a whitish semi-opaque glairy substance of homogeneous consistence, while the walls of the canals have an opaque yellow tint, and are readily torn. The whole of the surrounding softer parts are converted into a gelatiniform substance, taking the place of muscles, the tendinous and fatty

FIG. 57.\*

structures being less readily changed. The foot presents externally the peculiar turgid appearance which it so often assumes in bad cases of scrofula. Besides the canals, pink stains or streaks are ob-

---

\* The figure represents the general appearance on section of the diseased foot in the fungus disease of India. It is based upon dissections, and on three sketches made immediately after amputations of the limbs (CARTER).

(a, a.) The *fungi*, some of which are globular and of large size, others smaller and more irregular, and others mere granules. The former are lodged in the spherical cavities in the bones. (b, b.) The *canals* in the soft parts and bones which lead to the free surface of the skin. They frequently communicate, and are lined by a continuous membrane; in them are contained the *fungi*. (In a diagram of this sort it is impossible to represent the soft glairy material which also occupies the canals.) (c, c.) The *apertures* on the surface where the canals terminate. They are often very numerous, and frequently in them may be seen impacted the black particles. (d, d.) The pink-colored *stains or streaks* in the skin, above described. They are common to both varieties of the disease, and by them it is supposed the growth is multiplied.

It is to this variety of the affection that the term "fungus disease," which correctly expresses its nature, was, *par excellence*, originally applied. Hitherto no other instances of it have been distinguished, except those described by Dr. Carter; hence it may perhaps be regarded as comparatively unfrequent. The fungus particles or masses are of a deep black color, and of firm consistence; they are sometimes as large and as round as a pistol-bullet.



The species has been named by Mr. Berkeley as *Chionyphe Carteri*, the name serving to record the labors of the two Carters "united in their love of science, though not in consanguinity." It is highly probable, as Mr. Berkeley observes, that many of our common moulds occasionally commence with a similar condition. The first indications of vegetation on tainted meat or paste assume the form of little gelatinous spots, of various colors, consisting of extremely minute distinct cells, and these seem to be an early stage of a common species of *Aspergillus* and *Penicillium*, or other genera. If there be any truth in the notion entertained by Mr. Berkeley, that hospital gangrene depends upon some vegetation of this nature, acting as a putrefactive ferment, there may be good reason for believing that the red spots in question are really the commencement of the disease under consideration.

In the second form under which the disease appears the black fungous masses are entirely wanting, and in their stead masses are found of what looks like sloughing tissue. White granules, however, occur in the cavities and in the discharge, which appear to be a form of the same fungus, though the identity has not been proved. Under the microscope it wears the appearance of a congeries of large cells filled with smaller ones. Whether the perfect form of the plant be the same or not, the phases of the disease produced by it are exactly the same, and the malady admits of no other remedy than amputation of the foot.\*

A third form of the disease is known under the name of the *Madura foot*, from its having occurred at Madura. In this case the foot becomes enormously enlarged about the instep, though not so much at the ankle, while the toes are hypertrophied, and almost lost or embedded in the mass. The small bones are nearly destroyed, leaving behind a pallid or reddish tissue, while the others are more or less excavated. There are the same canals and external sanious apertures. In some parts they are filled with the same fleshy tissue, in others lined with it, where large cavities are formed by the junction of several canals containing broken-up osseous tissue from the exposed bones around, gray fragments, and masses of pigment. The pink color is partly owing to a general diffusion of pigment, which tinges the oil-globules, and partly to the presence of very numerous single or aggregated elliptic particles. These granules are from the  $\frac{1}{80}$ th to the  $\frac{1}{10}$ th of an inch in diameter, and occur sometimes as single ellipses, sometimes as two combined at the extremities of their major axis, and sometimes as square bodies

---

\* In the *second variety of Mycetoma* we find three or four different kinds of particles: these, however, are always light-colored and soft, and generally very small or minute. Of them only one form, certainly the more common, has been noticed by writers: it is that in which each particle is seen to be invested by a crystalline coat. The truly fungus nature of the more common kinds of granules or particles, and of that striking instance of the disease from Madura, is as yet only matter of inference.

It seems desirable that every step in the investigation of this disease (the elucidation of which is committed, as it were, to the medical officers of India) should be based on direct and repeated observation, and that speculation be refrained from as being at least useless.



within them. How the spores are carried there is at present a mystery, which may some day be cleared up, as the origin of many

FIG. 59.\*

intestinal worms has been, which can no longer be brought forward as an argument for equivocal generation.

[The fungi, the sole cause of the disease, are described by Dr. Carter, in his more recent publication on the subject, as follows :

(1.) The most common variety consists of small masses, of cheesy consistence and light-brown tint, formed of an aggregation of granular particles, and occupying the "loculi"—branching tubular canals passing off from the spherical cavities hollowed out in the osseous cancellous tissue. The granules or particles are visible to the naked eye, and resemble poppy-seeds; their number is immense, and they are freely discharged by the sinuses. Each consists of minute, rounded, or angular bodies (diameter about  $\frac{1}{60}$ th in.), which are enveloped on all sides by a deep crystalline fringe (stearic?), and which appear to be structureless, or only finely granular; they are degenerated fungi, and in their interior may sometimes be seen clear nucleus-like forms, which somewhat resemble spores, but which are probably oil globules.

(2.) The black fungus occurs in more or less spherical masses, attaining the size of half an inch in diameter; outer surface of a jet-black color,

---

\* Fundamental cells of the *Chionomyces Carteri* developed from the fungus foot of India, budding like the receptacle of an *Aspergillus* (BERKELEY and CARTER).





[Dr. Carter (*loc. cit.*) states that he had received word (1863) from an intelligent medical man, that he thought he had eradicated the disease by the free use of strong nitric acid.]

In some cases it would seem as if the foot had been in a diseased state when the fungus was introduced ; at least, the history of one case, which apparently commenced with a boil on the instep (which was treated by native doctors, a thorn being used several times as a lancet), indicates a lesion such as might well encourage the growth of a fungus parasite.

It is more than twenty years since surgeons in India first took notice of this affection of the foot in their official reports ; and one of the earliest to notice the disease was Dr. Colebrook, of Madras, then Zillah-surgeon at Madura, where the endemic character of the malady was first recognized by the term "Madura foot." An interesting account of the disease was afterwards published by my friend Dr. G. R. Ballingall, who was the first to describe the microscopic peculiarities of the disease, and he was led at once to distinguish at sight the tumor of the foot from any simple scrofulous affection, and to detect the prominent features by which he recognized the fungus foot as something *sui generis*. "Cases of diseased foot," peculiar to certain parts of the Bombay Presidency, were recorded by Assistant-Surgeon Bazunjee Rustomjee, in the fifth vol. (N. S.), p. 230, of the *Transactions of the Medical and Physical Society of Bombay*, and which Dr. Carter considers are "the most fully and carefully recorded instances of the 'fungus disease'" which had been published at the time Dr. Carter wrote his report. Most of the cases belong to the second form of fungus described in the text ; and practically the disease is regarded in India as a species of *caries*.

[Some idea of the frequency of the disease may be gained from the fact that individual observers in India reckon their cases by the score. Dr. Carter says that one person sent him particulars of seventy-five cases he had treated, and that even in Bombay, where the disease is not endemic, a year seldom passes without three or four cases being seen at the hospital. Other noteworthy features are : it has mostly a single local manifestation ; it is much more frequent in men, and during the middle periods of life, and commonest amongst the agricultural class ; it is not hereditary, nor peculiar to any diathesis (CARTER).]

**Symptoms.**—In the first variety the general form of the foot is oval, being much enlarged about the ankle and over the instep. On either side of the ankle-joint, on the dorsum of the foot near the toes, likewise on the sole, are numerous small soft swellings or tubercles, as large as a pea or marble, having pouting, puckered apertures, leading to fistulous canals ; and the skin surrounding these apertures appears lighter in color than elsewhere. (See specimens in the Museum of the Army Medical School at Netley.) The canals sometimes lead directly to the bone ; and a discolored glairy fluid, which exudes from the canal, sometimes carries with it a few black, gritty particles. The toes are distorted and displaced upwards,

Such a condition  
... twelve years; and the  
... exhaustion of the vital  
... forms are similar to

... by maceration, are  
... at once suggests  
... at work to pro-  
... the seat of cavi-  
... perfectly so. These  
... of a pin's head to  
... are formed by

... into each  
... not only so, but  
... directly or  
... superficial ones  
... like mere  
... a regular  
... the channel  
... sometimes  
... state the  
... collec-  
... to lead  
... the pe-

... disease  
... carries; the  
... appearance of the  
... which may be added  
... disturbance, pain,  
... scrofulous or syphilitic  
... commonly a certain test of  
... may usually be detected  
... glance will be suffi-  
... compared to mustard  
...

... (COLEBROOK).

... and studded  
... or less globular.  
... only, in which  
... themselves,  
... The disease  
... first sight re-  
... begins some-

... the appear-  
... of which  
... depressed

spots of one-third to one-half inch, or more, in diameter. The superficial dark layer of cuticle is cast off, leaving a very regular circular white surface, the centre of which presents a depression, closed at the bottom by a brownish layer, very thin in the middle. It was found on section that a small cavity existed beneath this depressed spot, or a tubular prolongation was detected running down through the remaining thickened cuticle and cutis into the subcutaneous cellular tissue, where it was not difficult to find the fungus particles, pink or yellowish-colored, and also in the cavity above named, in the superficial part of the cutis, or even on the surface of the latter, —the cuticle being raised.

Another and smaller specimen most clearly showed the development of the *fungi* at the very spot where, in all probability, their germs were first produced. In other parts there is a prolongation of the growth into the subjacent tissues, and there pink-colored particles were to be seen. The local nature of the whole affection—its very beginning—was here unmistakably displayed; and the superficial appearance of the skin gave the impression that a vesicle or blister had once existed there, not at all unlike that left after a *Guinea worm* has begun to discharge, as it is well known that the end of the worm makes its appearance in the centre of such a circular spot.

A further examination of the fungus particles showed their perfect resemblance to those of older specimens, and bodies not unlike spores were occasionally seen.

[It may be regarded as certain, that the hand or foot becomes accidentally inoculated with the spores of some fungus, which, at certain periods of the year, most likely during the wet season, makes its appearance on the soil of particular localities. The naked, unwashed feet of the agricultural laborer must be peculiarly liable to receive it, and the pre-existence of an abrasion of the skin is not necessary, for the spores are abundantly capable of passing into natural apertures—*e. g.* the sweat-ducts. The pinkish streaks on examination have been found to contain numerous spore-like cells in various states of growth, and probably constitute the first stage of the disease (CARTER).]

and the  
has been  
natural  
powers.  
those at

The  
of such  
the con-  
duce the  
ties men-  
cavities  
that of  
open ca-

From  
other, p-  
every ca-  
indirect  
some p-  
round l-  
tunnel.  
of com-  
equal t-  
sinuses  
tions of  
down to  
cular /

[Any  
could in-  
size of t-  
sinuses,  
the absc-  
or hect-  
taint.  
the nat-  
with th-  
cient, a-  
or pop-

The

The  
with ne-  
The wi-  
latter c-  
general  
seldom  
semble-  
times

Desc  
ance  
is ma-









**LANE MEDICAL LIBRARY**

**To avoid fine, this book should be returned  
on or before the date last stamped below.**

--	--	--

...ken. Sir William.  
...practice of



[Faint, illegible text, possibly a list or table of contents, consisting of several lines of small, spaced-out characters.]



